VOL. 15 OCTOBER, 1941 No. 4

THE PSYCHIATRIC QUARTERLY

PUBLISHED BY AUTHORITY OF THE
NEW YORK STATE DEPARTMENT OF MENTAL HYGIENE

The Psychiatric Quarterly, formerly the State Hospital Quarterly, is the official organ of the New York State Department of Mental Hygiene.

Volumes begin with the January number. Annual subscription rate, $2.00 in U. S. and its possessions; $2.50 elsewhere.

Editorial communications, books for review and exchange should be addressed to the editor, Dr. Richard H. Hutchings, Utica State Hospital, Utica, N. Y.

Business communications, remittances and subscriptions should be addressed to the State Hospitals Press, Utica, N. Y.

Entered as second-class matter April 17, 1917, at the postoffice at Utica, N. Y., under the Act of March 3, 1897.

DYNAMIC-CONCEPT TEST: QUANTITATIVE MODIFIED PLAY TECHNIQUE FOR ADULTS

BY A. NORDEMAN MAYERS, M. D., AND ELISABETH B. MAYERS, B. A.

Psychoanalytic theory has always suffered from a serious weakness—the difficulty one has in verifying and reinforcing it with simple, factual experimental data. This paper is a preliminary report dealing with a newly devised technique which may contribute some of this much-needed data. Only one of the many factors gained from our material, and only one class of patients, schizophrenics are presented. The report is presented now because the writers believe the results are unusually significant and characteristic. The intent is to demonstrate the amount of energy and direction a schizophrenic will exert when requested to function verbally on an accepted psychosexual level (we refer to such levels as oedipal, homosexual and heterosexual).

Play techniques have been widely used for child therapy. They have been utilized as a means of bringing out the phantasies and unconscious desires of the child.[1] Two major forms, with variations, have been evolved. One form is a standardized or controlled technique where the child is put into planned play situations. This method has been developed by Conn,[2, 3, 4] Levy[5, 6, 7] and Solomon.[8] The form devised by certain other therapists—Gitelson,[9] Allen, Gerard,[10] Rogerson,[11] Blanchard[12]—consists of free or spontaneous play with little or no direction from the psychiatrist. Although play techniques have been used for children, they have rarely been applied to adults. However, Rosenzweig and Shakow[13] obtained interesting results from adults by using dolls and toy furnishings in a planned play situation in order to watch the behavior, record incidental productions and also obtain each patient's own description of his purpose and conceptions. Many of their findings are similar to those of the present writers.

Procedure

For the procedure, a standardized form of play technique is used. Five stories are asked for in the following order: (1) *A Story About a Child of Five or Under;* (2) *A Story of An Ideal*

Family; (3) *A Story About Best Friends;* (4) *A Romantic Story;* (5)*A Story About a Hero.* Although the material gained from all the stories is similar, *A Story About a Hero* was often more productive for certain patients. It also offered a chance for the schizophrenic to verbalize his fantasies more easily.

The topics of the stories were chosen because of their conflict content and also because they seemed likely to bring out the patient's conceptions of the major psychosexual divisions. Because of the impersonal presentation of the subjects (through the medium of the word "story" and the use of the indefinite article "a"), it was hoped the patients would be lulled into talking about their conflicts and airing their hostilities. The results have more than justified this hope. *A Story of a Child of Five* is intended to bring out the oedipal and preoedipal preoccupations of the patients. *A Story of An Ideal Family* should call forth many of the oedipal conflicts and also bring out sibling rivalry. *A Story About Best Friends* hints at the latent or overt homosexuality of the patient; and *A Romantic Story* should yield much in bringing out the degree of heterosexual adjustment reached by the patient. Lastly, *A Story About a Hero* tends to bring out material, the ultimate significance of which is still uncertain. It is composed of productions which apparently escape the censor more readily than in other stories; and in the productions of the schizophrenics, it ranks with the story of a child of five in bringing out the narcissistic phases of the personality. However, the stories do not necessarily produce material of the level suggested by the titles. Often one topic will perseverate through many of the stories, in spite of the change of subject indicated by the title. In some cases, patients will even change the title to suit the subject matter about which they want to talk.

The analysis of the stories shows much that is of interest; and this material will be brought out in the future. In this report, only the ability to tell a story which is appropriate to the title and which solves the created situation adequately and constructively will be considered. However, it may be said at this time that the results of the tests vary with regard to the individual personality, the type of illness, and the severity of the disorder. Notwithstanding individual variations, the tests have been found to follow fairly

characteristic patterns, according to the mental disorder. We are now in the process of converting into analysis-charts, which deal with many factors besides the one taken up in this report, over 100 records of cases chosen at random at the New York State Psychiatric Institute and Hospital.

The stories were obtained in the following way. The patient was introduced to the examiner in an interviewing room, or some place away from interference, and was asked to have a seat, although if he preferred to walk around the room, no restriction of activity was imposed. A cigarette was offered to put the patient at ease. He was told:

"This is something I would like you to do. I want you to make up some stories, very short stories, and I will give you the titles. You just make up a story about what I give you; but please talk very slowly so that I can take down what you say. Would you please tell me a story, just a *short* story, about a child of five or under?"

If the patient did not respond to the request, various forms of coaxing were used; but the form which this assumed, depended upon the individual patient. The ultimate aim was to get the patient to produce. The results, as will be shown, have been gratifying in getting almost mute schizophrenic patients to produce at great length. Some patients required flattery, others cajolery and reassurance. If the patient was often reassured, told that he was doing well, the results were better. No set form of instructions was given any patient. The purpose was to have the patient make up stories, and any fair method was employed to get him to do just that. The order in which the stories were given was fixed, except when a patient blocked, in which case the next in order was offered, with a return later to the blocked story. In almost all cases, the blocked story was then obtained. The stories may be taken by the physician or by a stranger to the patient. It was found that the tests taken by a stranger yielded more material. The complete set of stories required about 30 to 40 minutes. However, they varied and might require a much shorter or a much longer time. Occasionally, the story was interrupted if the productions were too lengthy or disconnected.

TABLE 1. 33 SCHIZOPHRENIC PATIENTS

Title of story	Dysfunction	Partial function	Function
Child of five			
Hebephrenics	6	3	3
Catatonics	2	2	2
Paranoids	3	5	3
Simple	0	3	1
Totals	11	13	9
Ideal family			
Hebephrenics	2	7	3
Catatonics	2	4	0
Paranoids	4	7	0
Simple	0	4	0
Totals	8	22	3
Best friends			
Hebephrenics	9	2	1
Catatonics	3	3	0
Paranoids	8	3	0
Simple	3	1	0
Totals	23	9	1
Romantic			
Hebephrenics	12	0	0
Catatonics	6	0	0
Paranoids	8	1	2
Simple	4	0	0
Totals	30	1	2
Hero			
Hebephrenics	3	4	5
Catatonics	4	1	1
Paranoids	4	3	4
Simple	1	2	1
Totals	12	10	11
Grand totals	84	55	26

TABLE 2. 10 CONTROL SUBJECTS

Title of story	Dysfunction	Partial function	Function
Child of five	1	4	5
Ideal family	1	6	3
Best friends	1	1	8
Romantic	2	2	6
Hero	1	1	8
Grand totals	6	14	30

Data

Table 1 is the result of reducing to three simple categories every complete set of stories obtained to date from cases diagnosed as schizophrenics—a total of 33 charted here. The only stories taken from schizophrenics which do not appear in the chart are (1) from those who refused to perform or complete the test—three; (2) from those tests taken prior to the standardization of the titles—three; (3) from patients who gave repeated tests—two; and (4) from three cases where the diagnosis was in question.

The stories as classified in the chart were put in the following categories: "Function," "Partial Function" and "Dysfunction." All the categories were arbitrarily chosen; and in every case, the patient received the benefit of the doubt toward the side of function.

(A) *Function.* By this term the writers mean production of a story which is pertinent to the title and in which the patient creates a situation and solves it adequately and constructively.

(B) *Partial Function.* In this classification were placed those productions which had two characteristics—they were apropos to the subject matter, and they were descriptions, definitions or descriptive narratives. When a patient substituted a relevant descriptive dissertation in lieu of the requested story, it was included in this category of partial function, because it showed that the patient was able to comprehend and discuss the topic, even though he did not function completely.

(C) *Dysfunction.* This category contains all those productions which do not fulfill the stated requirements for function or partial function. In view of the limitations placed on categories A and B it was obvious that dysfunction would contain a great variety of type responses which seemingly had no connection with each other. However, their connection was manifest and basic. They simply did not conform to categories A and B and, therefore, had to be placed in category C. The type of responses included refusal to produce, changes in the title, unresolved plots, destructive endings, rambling dissertations, and presentation of an animal character in the title rôle.

Although the total number of cases is small, the writers would like to call attention to certain trends to be seen in the tables. Out of a total of 165 stories (33 cases of five stories each), 51 per cent fall into the category of dysfunction, with 33 per cent in partial function. Only 16 per cent of the schizophrenic stories charted actually fall into the category of function.

In total, there are only 11 dysfunctions in stories about a child of five or under, but this number of dysfunctions doubles in best friends stories and trebles in the romantic stories where it reaches 90 per cent. The figures for the ideal family and the hero are taken up later.

Partial functions in each group are child of five, 13; ideal family, 22; best friends, 9; romantic, 1; hero, 10. These figures are more or less constant except in ideal family and romantic stories. In the former, the number is almost double the others and is due to descriptions and philosophizing as to what constitutes an ideal family. Note that in this category of ideal family stories the number of dysfunctions is at its lowest ebb and that the number of functions has taken a sudden drop, both of which contribute to this dramatic upswing of partial functions. In the romantic stories, the partial functions, with one exception, have become complete dysfunctions.

Functions in each group are child of five, 9; ideal family, 3; best friends, 1; romantic, 2; hero, 11. Around one-third of the schizophrenics showed the capacity to function when the stories involved a child or a hero. In the hero stories they showed the greatest number of functions. Apparently, this type of story allows the greatest latitude in fantasy and implies little or no interpersonal attachments. However, the schizophrenics failed to function except for a few cases when interpersonal attachments are required.

The table of schizophrenics stands in marked contrast to the table of normals. In the grand total of normals, Table 2, they show three-fifths functions as against 16 per cent for the schizophrenics; and, whereas the schizophrenics show 51 per cent dysfunctions, the normals show 12 per cent.

The writers would like to call attention to the varied reactions of the different types of schizophrenics as brought out in Table 1. Further studies will be made in this regard.

Various types of productions elicited are now quoted:

Stories of Child of Five or Under

K—Female No. 63. (Hebephrenic schizophrenia.)

They're mostly in the park. They get wheeled about in their baby carriage. The mother or nurse takes them out and sometimes wheels them around and sometimes she sits on a bench with the baby carriage next to her. They go to—they go out to the park in the morning and in the afternoon and—one second—just let me think awhile—and they go to bed at 6 o'clock at night and they wake up at 6 o'clock in the morning and they take a two-hour nap in the morning in their baby carriage outside from 10 to 12—no I made a mistake—a three-hour nap in the morning and a two-hour nap in the afternoon from 1 to 3 and often from 2 to 4. As the child grows older he takes a two-hour nap in the morning from 10 to 12 and a two-hour nap in the afternoon from 1 to 3 or from 2 to 4, and finally when the child gets to be three years old he—he just sleeps I think 12 hours at night—yes just 12 hours at night with maybe a two-hour nap in the afternoon from 2 to 4 and that's all. I made a mistake—a slight mistake. When he gets to be three they have those lying down things in the baby carriages. When he gets to be three—at the runabout stage. Before he gets to the runabout stage they have the lying down baby carriages. When he gets to the runabout stage, they have different kinds of carriages—the kind he sits up in and I think then at this period he just sleeps 12 hours at night and may take a nap in the afternoon before going out to the park and then later on at the runabout stage he just sleeps 13 hours at night and doesn't take any nap—he just goes out in the air in the different kind of carriage—the sitting up kind. He walks about and runs about at the runabout stage which is a year and two years old and when he's three, he has no carriage at all he—he—just goes out with his nurse and plays with other children in the park and has no nap—just sleeps 12 hours every night from 6 in the evening until 6 in the morning. So I said he plays with other children in the park and he goes out to the park at 9 or 10 in the morning and stays out until 12 and then comes home for lunch and then goes out again in the afternoon at about 2 to 5 or 3 to 5 somewheres around there. (Wants to say more.) (Partial function.)

G.—Male No. 26. (Hebephrenic schizophrenia.)

(Repeats title.) Well, there were a group of children playing and they decided that they wanted to play house—in other words someone had to portray family life in other words one had to be mother, another the father —and another the son. There were two boys and two girls and then one boy wanted to display his ability more or less and didn't want to be a member of the family so he decided to be the handy man around the house. The other one who was younger and didn't understand much about how a house was kept decided to be the housekeeper, so that left the other two girls to decide what they wanted to be—mother, father, son—to complete the family circle. Well, the two girls more or less were keen on portraying the mother 'cause it was closer to their hearts. But that antagonized the housekeeper and the handyman because they needed someone to direct them. Well the girl—the girl of five or so—youngest in the bunch—sensing that more or less the mother was important wanted that most of all because she'd be able to dress the baby—the doll—and put it in the carriage and it struck her that being boys why didn't they want to be the father? So therefore the two girls were unable to reconcile themselves to play the father and prevailed on the boys, but the boys were keen on their job and setting the house in order and said to run. That left nothing interesting to do except take care of the baby and enjoy the fresh air while after some time the younger one came back and found that the housekeeper was giving all the orders on how to prepare the meal and the older girl, who was now the father, jealous of the little one playing the mother could not stomach the way the housekeeper and the handyman were absorbed in their work, prevailed on the little one to take the part of the father. But this she didn't care to do because it struck her now that both the handyman and housekeeper were interested in their work, were not able to see that merely taking care of the baby was sufficient that the housekeeping wasn't relegated to the background and then it struck her that after after all being the father wasn't as severe and brutal as she thought it might be, but that being the father meant that she was more or less out of the picture and did not exemplify the qualities she had come to know. She was left in tears with the job of coming home and watching the others busy themselves with trifling matters and then she spoke up and told both housekeeper and gardener there was no use in trying to keep the house ship-shape—hold of what was going on and the house in order, if there wasn't enough attention paid to satisfy both mother and father. For then there was something more to keeping house and fondling with the baby. And then when she began to speak up and order both gardener and housekeeper on how to do their

chores and other work, the older girl dropped the baby and just stood by watching her and then all awoke to the appreciation of the idea of being father was something more than being old or immature and slightly tougher than ordering the people about because they suddenly found they could not decide what would be a fitting job for a youngster of five to hold to be head of house so they sufficed with doing their job and listening to one another and the older girl compromised herself on the idea that running house was not without difficulty but not the severest of jobs. The younger girl, sensing that the others wanted their appropriate share of the housekeeping quickly spoke up and said if there's no job that's fitting she would take a story book and read it just like father did at home. The others, angered at the idea at the ease of running a household if it were orderly arranged could speak no more for she had exhausted all their arguments against having someone superior to do the ugly and necessary jobs in life. I think that ends it about there. (Partial function.)

Story of An Ideal Family

O.—Male No. 60. (Paranoid schizophrenia.)

An ideal family can go without any quarrels or disputes and—a—an ideal family is one that can get along without any quarrels. An ideal family is one where a man and wife love each other. I don't know why a man loves a girl—marries her and they have a family, have a baby and that's an ideal family.—Man trouble. (Chatter.) Does that pertain to marriage—an ideal family I mean? I can't talk because people are listening to what I'm learning. I learn a lot. But I don't learn from school but from life. (Partial function.)

M.—Female No. 59. (Simple schizophrenia.)

It is very difficult to state. It is very difficult to state the exact qualities of an ideal family. The exact qualities an ideal family possesses. To state exactly what qualities makes a family ideal, make a family, that make a family to—. It is very difficult to state what qualities an ideal family possesses. Harmony is an important factor that makes up an ideal family. Harmony is an ideal factor,—which contributes to—(So easy and now)—which contributes to the idealism of the family.

We'll start all over. The ideal family must have the following—I'll have to get a new opening sentence. (Long pause.) In my opinion the ideal family must possess the following qualities to—to—qualities to—in order to be rated ideal—that took a long time. Happiness is of prime importance. In order to achieve happiness each member must understand and consider one another. Arguments do occur but—but not continual discords and unreasonable attitudes towards one another. If the parents are in harmony

there is a better chance for the children to be in harmony with one another and with the parents and with their parents. If one parent disagrees with another about an issue concerning a child—the child—a child—is best that they can—that they come to a conclusion out of the child's knowledge, away from the child's presence and not to bring the disagreeing factors to—with it—to the child's sight or hearing. Now why? I have to think why (long pause). These—a—debates in the presence of the child would only confuse him and—and probably upset him to a degree of—of illness. Harmony for the sake of the children and the—and the parents, the parents should always act considerate and kind to one another so as to—to—instill these things into the children by example. One child should not be more favored by—by—by—by—having, by receiving more than the other members or by receiving special attention—more attention from the other members—more attention should not be shown more attention and love by—than the other members for this would give the other members—for this would arouse jealousy—this would—for this might—this might cause jealousy and discord amongst the members—and that would gradually be followed by arguments and fighting. (Partial function.)

Story of Best Friends

L. B.—Female No. 55. (Catatonic schizophrenia.)

Are there best friends? Do we have best friends? No. I don't think there is such a thing as friends—family—people—anything. If you were to choose a friend would you pick a man or a woman? (Dysfunction.)

S.—Male No. 102. (Paranoid schizophrenia.)

I'll tell you a short story—it bears a great connection to this. Well—some parts of my life, I dropped everything and actually went on the bum. When I'm poor no one has any designs upon my money or my life which used to be the case when I had money and position. In the last three years I figured I'm tired of travelling around—lived on WPA. In my travelling a little over three years I met a Polish fellow; he had the look of a murderer.—Finally one night in the end of March three years ago—it was a terrible night quite late, this person that I always avoided stopped me and asked me for something to eat. He was trembling, and I could see he was ready to fall down from famine and weakness. I took him to a cheap restaurant. When he asked me for a night's sleep, I did not know what to do. It was late around 1 o'clock. I lived in a very small room and a very small bed. I took him up. It took him about 25 minutes to make the steps—that's how weak he was. When he got into the room, he fell asleep and was sick three days. I hardly slept three days and went through Hell. I was afraid the landlady would come in—find I made a hospital of my room—

throw me out and lose my relief. I had the feeling he was going to die on my hands. On the fourth day he was better. On the fifth day he said he wanted to go to the Public beds because he was lousy, filthy and crabby. I gave him clothes and took a bed and he began to straighten up. He said, "Joe, I'm a person who don't talk much and I'm not a kind person. I'm a very hard and selfish person and I'm going to prove to you I can be different to a person who deserves a break." I said, "You want to give me a break. I say: Go away and just don't come back." He did come back. I was very much puzzled. At first I thought the man was a queer. I slept with him—I don't like men. I saw the man was the same. Suppose I tell it—and you put it in your words. He began to work and I told him to take an advance on his pay and let him take a room. He went and came and I couldn't get a word out of him. By the coming Saturday night he came home and put $12 on the dresser and invited me to the movies. I was so mad at him for the way he acted—I'll never act kind to any human beings—I'll spend it on birds—I didn't go to the movies. He left money—went to movies—came back next day, didn't work and began to talk. He told me, "I'm a very selfish person." He said, "I run my own life—had a good education—nobody could have any influence on me. For myself I'm just a bum, but I feel that if you are willing to take an apartment with me, that I can be good to you and thereby benefit myself." Well—I just let him talk me into it. Of course I thought the whole thing crazy and ridiculous. Even if my own person is involved and there is something new to learn I want to know the outcome. Did I say I took an apartment? I took the apartment and there was very little in it, and in a short while I fixed up a pretty decent apartment and we had half-decent clothes; it was a regular household and then in the beginning he found a job in the mountains as a cook. He came back with $350.00. He came back. He worked quite a bit in the city. Well, the fellow was still unkind and unsociable, of course, to other people who came to the house. He lost faith in everyone. He didn't talk much to me, but his behavior and treatment coming from such a person was marvelous. We had a place where we kept a couple of dollars for expenses—so I wouldn't have to worry. In short, this is the best friendship I really ever had. There is an ending, but that wouldn't be strong friendship. Maybe I should tell the end. Maybe we was still best friends. (The patient cries.) About 1939 he came back from the country and said he was going to buy a business and a lunch room and restaurant. Why did I have to tell you this story?—I could have picked—he was going to buy a lunch or restaurant and we were going to be partners, but before it was too early for the appointment, so we used to frequent the New Delancey Theatre. He left me in the house with $75. I have a hunch he never came out of the movies alive. (Dysfunction.)

Romantic Story

P.—Male No. 30. (Paranoid schizophrenia.)

(Pause) During the early years of our lives we are confronted—the majority of us are confronted—with some sort of romance. (Dysfunction.)

P.—Male No. 37. (Hebephrenic schizophrenia.)

A—(pause—sigh)—James—a—Peabody—a—and Lucy—Lucy Williams meet by accident—on a Southern cruise. They're both seated at the same table on a trip to Argentina. Both become very interested in each other and decide to come back on the same boat—a—They have a serious love affair on board which culminates in the birth of a child. They both take a trip to Chile the following year and became married, shortly before the child was born—a—Lucy does not tell what's his name Williams there—or Peabody—her husband that she is already married. Her husband finds out about their marriage—from the—a—captain of the ship who is a good friend of his—call him Mr. Williams—Mr. Williams finds out. He decided to put an end to the affair by—a—mur—murdering Mr. Peabody. When both of them come back from the trip, they find—Mr. Williams in their living room. Mr. Williams—a—shoots Mr. Peabody who—a—clubs—a—(He mutters.) Mr. Peabody clubs Mr. Williams over the head. Kills him. The case comes up before court and Mr. Peabody is acquitted on the basis of self-defense. The Peabodys rejoice on their union after its success O. K. (Dysfunction.)

Story of a Hero

C.—Female No. 100. (Hebephrenic schizophrenia.)

Hero? A hero lived in Greece—(She laughs for two minutes.)—And historical? Any hero, oh, and he was an Athenian and Spartans fought—(Again laughs.)—and he saved Athens. (Function.)

K.—Male No. 24. (Hebephrenic schizophrenia.)

Edward C. Charleston took a trip to North Woods to take a vacation. He wanted to do shooting—rifle shooting. There were many animals in the North Woods. Mr. Charleston loved to shoot, so on this spring day Mr. Charleston was dressed very splendidly in hunting clothes and was going through Khyber Pass when all of a sudden he came upon a gorilla, and Mr. McGregory was so astonished that his gun flew right out of his hand and the animal was just about to pounce on Mr. McGregory when another animal came and rescued him—the man. (Partial function.)

In taking these tests at random, the writers made the discovery that so-called non-productive, and in a few cases almost mute patients, would respond. Out of the 33 cases, seven patients were known to be comparatively non-productive, a few practically mute.

As has been pointed out before, the total partial functions of the schizophrenics are more than double those conforming with the writers' definition of function (33 per cent against 16 per cent). These results tend to reinforce the theory that the schizophrenic either does not possess, or else cannot sufficiently focus or direct, affect-energy to function adequately in a situation necessitating the utilization of both emotional and intellectual abstract thinking. The writers hope at a future date to elucidate their reasons and their material for making this differentiation between two types of abstract thought.

Summary

A standardized play technique has been devised for adults and has been used on 33 schizophrenics and a group of control subjects. It requires spontaneous stories in response to five titles chosen for their psychosexual and conflict content. These stories were *A Story About a Child of Five or Under, A Story About an Ideal Family, A Story About Best Friends, A Romantic Story, and A Story About a Hero.*

In a series of 33 cases the dysfunctions were 11 in child of five, 8 in ideal family, 23 in best friends, 30 in romantic and 12 in hero. The dysfunctions doubled from childhood stories to friendship stories and trebled in romantic stories. Reasons for the smaller amount of dysfunctions in stories about an ideal family and hero, were given.

Partial functions in the stories were 13 in a child of five, 22 in ideal family, 9 in best friends, 1 in romantic and 10 in hero stories. In this series the most significant findings were the number of partial functions in the category of an ideal family and the almost complete loss of partial function in romantic stories. The former finding shows the tendency of the schizophrenic to give a descriptive-philosophical concept, and the latter dramatically reinforces the impressive number of dysfunctions in the romantic stories.

The so-called functions were few in the schizophrenics and stand in marked contrast to the table of the normals. There were 9 functions in the child of five, 3 in ideal family, 1 in best friends, 2 in romantic and 11 in hero stories in the schizophrenic table.

Keeping in mind the effort of the foregoing report to maintain a dispassionate and investigatory attitude, the opinion is expressed that the table of schizophrenics presented here offers significant experimental proof of the theory that schizophrenics in telling a story will tend to function best in a purely narcissistic environment and that the capacity to function decreases as the more mature levels of psychosexual development are reached.

If further experiment substantiates the data herein elucidated (and other material to be written up later), this dynamic-concept test will reinforce psychoanalytic theory by simple, factual experimental data.

ACKNOWLEDGMENT

The writers wish to express their gratitude for the help and cooperation given them by members of the staff of the New York State Psychiatric Institute and Hospital.

Department of Psychiatry
New York State Psychiatric Institute and Hospital
New York, N. Y.

REFERENCES

1. Allen, F. H.: Therapeutic work with children. Am. J. Orthopsychiat., IV, 4: 1934.
2. Conn, J. H.: A psychiatric study of car sickness in children. Am. J. Orthopsychiat, VIII, 1: 1938.
3. Conn, J. H.: The child speaks to the psychiatrist: an introduction to the method of play interview. Occup. Ther. and Rehabil., XVII, August, 1938.
4. Conn, J. H.: The play interview: a method of studying children's attitudes. Am. J. Dis. Child., 58, December, 1939.
5. Levy, D. M.: Use of play technic as experimental procedure. Am. J. Orthopsychiat., III, 3: 1933.
6. Levy, D. M.: Hostility patterns in sibling rivalry experiments. Am. J. Orthopsychiat., VI, 2: 1936.
7. Levy, D. M.: Release therapy in young children. Psychiatry, I, August, 1938.
8. Solomon, J. C.: Active play therapy. Am. J. Orthopsychiat., VIII, 3: 1938.
9. Gitelson, M., et al: Clinical experience with child therapy. Am. J. Orthopsychiat., VIII, 3: 1938.
10. Gerard, Margaret: Case for discussion at the 1938 symptosium. Am. J. Orthopsychiat., VIII, 1: 1938.
11. Rogerson, C. H.: Play Therapy in Childhood. Oxford University Press, London, 1939.
12. Blanchard, P.: 1937 case for symposium and symposium. Am. J. Orthopsychiat., VII, 3: 1937.
13. Rosenzweig, S. and Shakow, D.: Play technique in schizophrenia and other psychoses. Am. J. Orthopsychiat., VII, 1: 1937.

A CRITICAL ANALYSIS OF INSULIN THERAPY AT ROCHESTER STATE HOSPITAL*

BY WILLIAM LIBERTSON, M. R. C. S. (England)

With the advent of the extensive use of insulin in the treatment of schizophrenia some workers prophetically foresaw the need for adequate control estimations before a logical evaluation of the rôle of insulin could be determined. Cheney and Drewry[1] in 1938 reported the results of "non-specific" treatment in 500 cases of dementia præcox. After following these cases for a period of from two to 12 years they found that 59 per cent were unimproved and 41 per cent showed some degree of improvement. They, furthermore, concluded that if the patients had shown symptoms for a period of not more than six months, then one-half of them would be benefited by "non-specific" treatment. Hunt, Feldman, and Fiero[2] in the same year studied 641 cases and found a total improvement rate of 35.1 per cent. In their analysis of 161 cases of less than six months duration, they found a total improvement rate of 54.6 per cent. Malamud and Render[3] examined 309 "untreated" patients who had been discharged from the Iowa State Psychopathic Hospital. Of these, 177 were followed for five years or more. At the end of their period of observation Malamud and Render found that 58 per cent were unimproved, 32 per cent showed varying degrees of improvement, and 10 per cent were dead.

With these results as a partial basis for comparison, a study was instituted of all patients treated with insulin at the Rochester State Hospital since 1937. These numbered 190. Of that number, 13 were diagnosed in categories other than dementia præcox; and an additional 12 had received both insulin and metrazol. These were not considered in the survey. The remainder numbered 165; and they were studied for a minimum of seven months and a maximum of three years and nine months. As a further control, 165 additional patients were examined. They were admitted to the Rochester State Hospital during the insulin era, were diagnosed dementia præcox, but did not receive insulin therapy. These patients

*Read at the up-State interhospital conference at Utica State Hospital, April 25, 1941.

were chosen at random by one of the stenographers who was merely instructed to select a series of non-insulin-treated patients from the hospital admission book.

Table 1 shows the condition of the insulin-treated patients at the time treatment was terminated and their condition in January, 1941. It also shows the condition of the control cases in January, 1941, the latter having been followed for the same length of time as the insulin-treated group.

Comments Concerning Table 1

The classification used here is the one that is generally used in New York State hospitals and is more or less adhered to in other institutions. The recovered group includes those patients who have become entirely symptom free and who have developed insight. In addition, they must be able to adjust in the community

Table 1

Condition of patient	Insulin-treated patients at termination of treatment		Insulin-treated patients in January, 1941		Control patients in January, 1941	
Recovered	4 (2.4%)	62.4%	15 (9 %)	40%	8 (5%)	40%
Much improved ..	52 (31 %)		35 (21 %)		28 (17%)	
Improved	47 (29 %)		16 (10 %)		30 (18%)	
Unimproved	61 (36.9%)		95 (57.5 %)		93 (56%)	
Dead	1 (.6%)		4 (2.4 %)		6 (4%)	

at their prepsychotic level. The much improved group contains patients who are or should be symptom free but in whom insight is lacking or incomplete. Such a patient is able to adjust in the community at or near his prepsychotic level. The improved group contains patients whose symptoms have been incompletely alleviated. From observation, they should be able to make a better adjustment in or out of the hospital than before treatment.

The tendency to classify patients in one or other of the foregoing groups after a specific length of time is a tendency that has developed more or less since the advent of "specific" forms of therapy. Ordinarily, hospitals make no attempt to classify patients who have received no special form of therapy, until the patients have left the hospital. Consequently, due to our own lack

of foresight, it is not possible to compare the control group with the insulin-treated group at a time comparable to the time when insulin treatment was terminated. There is, however, no particular value in making such a comparison, since, as can be seen from Table 1 in the first statistical column gives very little information. Since the interest is in the ultimate outcome of the patients only columns two and three are of importance. That column one bears very little resemblance to the others can be seen at a glance. Furthermore column one reveals the uncomfortable fact that the definitions given have not been adhered to rigidly. The recovered cases, for instance, have not been followed sufficiently to know whether they will adjust at their prepsychotic level. The same criticism applies to the much improved group; and, by the same token, one has no indication of the length of time that mere improvement will last.

All of these unknowns can be eliminated only by examining and classifying the patients after carefully following their posttreatment course for an adequate period of time. When this is done, it is found that the initial improvement rate drops from 62.4 per cent to 40 per cent and that this is identical with the total improvement rate of the untreated cases. This is an astonishing finding because the control group was largely made up of patients who were deemed unsuitable for insulin therapy because of one or more factors. These factors were primarily age, duration of psychosis or evidence of clinical "hopelessness." Some patients were unsuitable for treatment because they began to improve too rapidly. The average age of the control group was 37, whereas that of the insulin-treated group was 28. The average duration of psychosis of the control group was nine years compared to 2.4 years for the insulin-treated group. It is difficult to know what to conclude from this. The superficial interpretation seems to be that an aged, non-insulin-treated schizophrene whose psychosis is of nine years duration has nearly as good a prognosis as a young, insulin-treated schizophrene whose psychosis is of only 2.4 years duration. It might be argued that 30 of the control patients fall into the improved group whereas only 16 of the insulin-treated patients are in this group. The very nature of the control group makes this an almost predictable finding. Furthermore, of these 30 patients,

25 are at home and only five remain in the hospital, whereas no less than seven of the insulin-treated patients in the improved group still remain hospitalized. It must also be remembered that in this survey the writer has not included the 12 patients who received both insulin and metrazol. These patients are obvious "insulin failures," and, in fact, only two of them left the hospital with any degree of improvement.

Although the second column in the table gives a more reasonable picture of the true state of affairs, it is by no means beyond criticism. In the first place, it represents the condition of the patients in January, 1941 and the policy of examining patients on a specific date is inadequate because it fails to indicate everything that happened during the preceding 12 months. To use a specific example we have many patients whose clinical course following treatment is indicated by the short-hand formula M - R - I - U. This means, of course, that the patient at the termination of treatment was much improved, one year later was considered recovered, during the next year had slumped to the improved status and at the end of another year was unimproved. It has not been unusual to find that, due to a particularly fortuitous set of circumstances, the patient may have an uninterrupted string of M's on his card because of the fact that for a time prior to each examination day he actually was much improved. Many of these patients have had acute psychotic episodes, for instance, in the summer months and at the end of the year have presented a normal appearance. In order to discover wherein the paradoxes exist it is necessary to examine each case in each of the groups longitudinally. Minute examination of this sort also helps in determining whether there is an actual cause and effect relationship between the treatment and the end result. Rosenberg and Moersch[4] raised this question in 1938 but made no attempt to answer it on the basis of only 33 patients studied. Halpern[5] raised a similar question when she expressed doubt that remissions in certain cases with protracted treatment or late remissions after shock therapy could be positively considered the result of shock therapy. The following analysis of individual cases helps to clarify some of these issues.

The Dead Patients

Only one of these patients died during the course of insulin treatment. She was a young catatonic who had not reached a coma dose. Following the fifth day of treatment, she developed acute nephritis and died in uremia. The second patient, after making what appeared to be a complete recovery, committed suicide within two months of the termination of treatment. She was diagnosed dementia præcox simplex. The third and fourth patients were unimproved following treatment and died of "natural causes" five months and nine months, respectively, after treatment.

Comments concerning this group on the whole are not particularly pertinent to the general subject matter of this paper. Since one of the "recovered" patients committed suicide, however, there should be a few words about the four patients originally classified as recovered. In the experience at Rochester, this classification, while being very rational and desirable, is nevertheless unjustified when applied prematurely. Of the four cases originally called recovered, the average duration of remission was less than five months. In addition to the patient who committed suicide there were three cases who lapsed back into an obviously psychotic state six months, two months and nine months, respectively, following the termination of treatment. This indicates that in spite of a very promising clinical picture it was very premature to apply the label "recovered" to each of the four patients.

The 15 *Patients Classified as Recovered in January,* 1941

Eleven patients of this group follow:

Name	Diagnosis	Duration of psychosis	Duration of uninterrupted remission
KVV	DPC	2 months	11 months
VP	DPC	2 months	11 months
JG	DPC	4 months	11 months
MM	DPP	6 years	3 yrs. 9 mos.
JLB	DPP	8 months	3 yrs. 9 mos.
JW	DPH	12 months	2 yrs.
ED	DPC	4 months	2 yrs.
TES	DPH	6 months	1 yr. 9 mos.
EF	DPH	6 months	1 yr. 9 mos.
AMM	DPP	5 years	14 months
MEF	DPC	2 months	9 months

The remaining four cases are abstracted at slightly greater length because of points of particular interest.

CED, dementia præcox, paranoid, of two years duration, after 43 treatments, was in November, 1937, considered much improved. He was paroled but within two months relapsed and was returned to the hospital. He had a second course of 45 treatments and on this occasion was considered merely improved. He subsequently went home, improved steadily, mentally and physically, and in January, 1940, and in January, 1941, was considered recovered.

AM, dementia præcox, catatonic, of five months duration, after 31 treatments, was considered in June, 1937, to be much improved. He was paroled in July, 1937, but within two weeks relapsed and was returned to the hospital. In October, 1937, he was again paroled but within six weeks was returned to the hospital. Thereafter, for nearly a year, he was mute and had to be tube-fed. He then spontaneously began to improve, was paroled in November, 1938, discharged in November, 1939, and since then has been working steadily. Clinically he appears to be recovered.

NKG, dementia præcox, paranoid, of five months duration, began to improve several weeks before insulin treatment started. Consequently, by the time he had had only 13 treatments, he appeared well. His condition was much improved in November, 1937. He was paroled, a year later was discharged as recovered and clinically he is still recovered.

AVK, dementia præcox, paranoid, of eight months duration, after 55 treatments, in January, 1938, was considered merely improved. Within a short time, she became mute, was destructive to clothing and had lapsed back to her pre-insulin state. One year following treatment, she began to improve; and, in January, 1939, was paroled, made a satisfactory adjustment and, in January, 1940, was discharged as much improved. Her condition in January, 1941, places her in the recovered group.

A study of this group of cases indicates that in the first 11 it is difficult to question the rôle of insulin. In the remaining four, however, it is equally as difficult to establish satisfactorily a cause and effect relationship. Halpern's statement, already cited, pertinently applies to three of these cases.

The 35 Much Improved Cases as of January, 1941

As in the previous group, some cases are found where it is again difficult to question the rôle of insulin, since the improvement seemed to occur during the course of therapy and was subsequently

maintained. These numbered 21. The remaining 14 are abstracted briefly because once again they cast doubt on the rôle of insulin.

DR, a dementia præcox, hebephrenic, of three months duration, had 49 treatments and in November, 1939, was considered much improved. Six months later she had another acute hebephrenic exacerbation indistinguishable from the first one. She recovered spontaneously and, in January, 1941, was placed in the much improved group. She is now on parole.

JE, a dementia præcox, paranoid, of seven months duration, after 41 treatments was unimproved in that he was still very confused, assaultive and hallucinated. For 16 months, he was on a disturbed ward. Then he suddenly improved and, in January, 1940, was placed in the much improved group. He has since maintained his improvement.

CB, a dementia præcox, catatonic, in 1933 had a psychotic episode following which he had a remission lasting six years. Following a new episode, 52 treatments were given, ending in March, 1940. He was considered merely improved. In spite of the fact that he still harbored ideas against his father, he was paroled in June, 1940, and in August, while at home, he began to improve very dramatically. His genuine improvement thus appeared five months after treatment terminated.

MJC, a dementia præcox, catatonic, of two months duration, after 71 treatments in January, 1940, was considered unimproved. Two months after treatment stopped, she began to improve. She was paroled in March, 1940, and is still in satisfactory condition.

FD, a dementia præcox, paranoid, of four months duration, after 26 treatments in December, 1937, was considered unimproved. He was a voluntary patient and discharged himself from the hospital. During his year at home, he gradually lost his delusions and since that time has steadily been a much improved patient.

GEN, a dementia præcox, hebephrenic, of two months duration, had 29 treatments ending in October, 1937. He was considered much improved. He was paroled, relapsed completely, in April, 1939, was returned to the hospital and within six months spontaneously regained his former status. He was again paroled in September, 1939, and is still out of the hospital in satisfactory condition.

EAW, a dementia præcox, catatonic, of 10 months duration, after 41 treatments in January, 1938, was considered improved, although still dull and simple. He was paroled in March, 1938. Twelve months after his treatment ended, he suddenly gained spontaneity, improved in all respects, went to work and since then has been placed in the much improved group.

ON, a dementia præcox, catatonic, of six months duration, after 48 treat-

ments in March, 1938, was considered unimproved. Two months later, she rapidly and dramatically began to improve, was paroled shortly afterward and since then has remained in satisfactory condition.

WM, a dementia præcox, paranoid, of three months duration, after only 16 treatments in September, 1938, was considered much improved. He was paroled but one month later completely relapsed. A second course of treatment in November, 1938, was interrupted after 12 days because of a prolonged coma. The patient was left with definite organic brain damage which has persisted to this date. Six months following the prolonged coma he showed enough "mental improvement" to be paroled and since that time has been working and is considered much improved.

AB, a dementia præcox, hebephrenic, of three years duration, after 31 treatments in April, 1938, was unimproved. She was paroled against the judgment of the clinic psychiatrists; and at home her condition fluctuated markedly, at times being actively psychotic and at other times being moderately rational. As of January, 1941, she is much improved and is working and for the time being is apparently normal.

LS, a dementia præcox, paranoid, of eight months duration, after 43 treatments in June, 1938, was merely improved. Six months later she was asocial, silly and confused. In March, 1940, 21 months after treatment, she suddenly improved, was paroled, got a job and as of January, 1941, is much improved.

HE, a dementia præcox, paranoid, of one year's duration, in December, 1939, after 77 treatments was improved, although he was still hallucinated. Nine months later, he completely lost his hallucinations. In September, 1940, he was paroled, obtained work and he is still working and much improved.

JR, a dementia præcox, catatonic, of eight years duration, after 55 treatments, in March, 1939, was considered improved. Improvement, however, was not sufficient to warrant parole, and 13 months elapsed before she showed sufficient further improvement to be returned to society. Since April, 1940, she has been well and has been working as a practical nurse.

LVR, a dementia præcox, catatonic, of seven months duration, after 55 treatments, in October, 1937, was much improved. He rapidly lost his improvement and it was not until two and one-half years later that he regained it sufficiently to warrant parole. He is now on parole and is getting along satisfactorily.

Perusal of these briefly abstracted cases reveals some patients whose improvement began to appear many months after treatment ended, other cases in which improvement followed a relapse and

one definite case in which improvement began before the course of insulin treatment started. It is obvious that many of these patients appear in the annual much improved group by virtue of the habit of classifying patients at stated intervals. It is seen that some of the patients improved many months after treatment was terminated. Ross[6] quotes Sakel as saying that such improvement is akin to a process of ripening. While it is difficult to argue dogmatically against such a statement, it is, nevertheless permitted to question if such "ripening" continues after a relapse has occurred. It is felt that it is also permissible to ask how long such a process of ripening can go on.

The Improved Group as of January, 1941

There were 16 cases in the improved group; and, of these, there were only six in which it seemed reasonably certain that there was a direct relationship between the insulin treatment and the patient's condition as of January, 1941. In the other 10 cases other factors must have been at play. These 10 cases are abstracted here.

CS, a dementia præcox, catatonic, of three months duration, had 17 treatments and, in May, 1939, was considered recovered. She was paroled in September, 1939, and was returned to the hospital unimproved in June, 1940. By September, 1940, she again had improved sufficiently to be sent out on family care and she is still out of the hospital.

CS, a dementia præcox, catatonic, of two years duration, following 50 treatments in January, 1940, was unimproved. He was paroled in February, 1940, still unimproved. At home he steadily improved and he was about to be discharged as much improved in February, 1941. However, between his penultimate and ultimate clinic visits his hallucinations returned to such a degree that the increased tension associated with them forced him to give up his job. This episode of hallucinosis lasted only one week. He remains out of the hospital on parole. Placing this patient in the improved group was a compromise dictated by the recent bout of hallucinosis. Under other circumstances he might have been placed in the much improved group.

MB, a dementia præcox, hebephrenic, of six years duration, had 41 treatments ending in June, 1940. She was unimproved, in that she was still

posturing and gesturing. Two months following treatment, she suddenly began to improve and in November, 1940, was paroled and is still out of the hospital.

DP, a dementia præcox, paranoid, of 12 months duration, had 70 treatments ending in May, 1940, with her condition unimproved. Two months after treatment stopped, she began to improve, was paroled and is still out of the hospital and getting along satisfactorily.

OK, a dementia præcox, paranoid, of three years duration, had 24 treatments, ending in October, 1937. He was considered much improved. He was paroled but returned from parole three months later actively psychotic. He had a second course of 37 treatments ending in June, 1938, and at this time was considered unimproved. In January, 1939, he escaped. He was discharged in January, 1940. He is classed as improved only on the basis of communications with his wife. Incidentally, this patient and one other escaped patient are the only ones in the entire series that the writer was unable to follow by personal interview.

MV, a dementia præcox, paranoid, of 15 months duration, finished 28 treatments in December, 1937. He was considered much improved. He was paroled in January, 1938, got along very poorly and was returned to the hospital in April, 1939. By October, 1939, he again improved enough to be paroled and he is still out of the hospital, his condition continuing to be improved.

HC, a dementia præcox, paranoid, of three years duration, finished 46 treatments in March, 1938. His condition was considered improved. He has remained out of the hospital since then. Examination in January, 1940, revealed that he was silly, superficial, scattered and delusional. He was therefore classified as unimproved on that date. Since then, although still delusional, his behavior is improved; and he is sporadically able to work.

MZ, a dementia præcox, paranoid, of five years duration, had 33 treatments, ending in May, 1938. He was improved but still delusional. After treatment, he immediately slumped back to his actively psychotic, assaultive state. He subsequently escaped in September, 1938, and since then he has been considered improved.

MW, a dementia præcox, paranoid, of five years duration, had 29 treatments, ending in March, 1938, and was improved. He was paroled in April, 1938, but within six weeks was returned in a mute and dazed state. Within nine days he got over his confusion and was again paroled. Four weeks later he was again returned to the hospital. In November, 1938, he was again paroled, but in three weeks was returned to the hospital. Within a

few days he once again improved, but because of his recent history, it was decided to keep him in the hospital. On February 4, 1939, he escaped and was discharged one year later. Communications from his wife indicate that he is still improved.

CM, a dementia præcox, paranoid, of four months duration received 21 treatments to May, 1938. He was then much improved. He was paroled in June, 1938, but 14 months later was recommitted. He had a second course of insulin, ending after 24 treatments in October, 1939, his condition considered improved. He lived up to the prognosis associated with the improved group and—within one month—lapsed back to an assaultive state and was sent to a disturbed ward where he remained for nine months. Following this period he again began to improve and, as of January, 1941, was once again in the improved group.

A brief analysis of the three groups abstracted in the foregoing, namely the improved, the much improved and the recovered, indicates that in finally evaluating the rôle of insulin certain cases should be excluded from the tables. If they are included, they carry with them the unfortunate suggestion that the therapy was responsible for the end result. In the groups mentioned above, it is probable that four of the ultimate recoveries were due to other factors, that 14 of the much improved were due to other factors and that 10 of the improved were due to other factors. If the writer may be permitted one more probability in what is already a long list of probabilities it is this, that the "other factors" involved are probably identical with the factors responsible for the improvements in the control patients. The doubtful cases, which have just been abstracted, add up to 28 which represents 17 per cent of the whole group and 42 per cent of the collective improved group. A revised table to indicate the cited probabilities would read as follows:

Favorable result probably due to insulin therapy	38	(23 %)
Favorable result probably due to other factors	28	(17 %)
Unimproved	95	(57.5%)
Dead	4	(2.4%)

Table 2 is a résumé of patients in the insulin-treated group and in the control group whose psychoses were of less than six months duration. There were 50 such patients in the insulin-treated group and 41 such patients in the control group.

TABLE 2

Condition of patient	Insulin-treated cases		Control cases	
Recovered	9 (18%)	60%	6 (15%)	61%
Much improved	17 (34%)		15 (36%)	
Improved	4 (8%)		4 (10%)	
Unimproved	19 (38%)		14 (34%)	
Dead	1 (2%)		2 (5%)	
	50		41	

The small number of patients in each of the two groups precludes any definite conclusions. However, the general agreement with the larger groups mentioned earlier in the paper, namely Cheney's 50 per cent total improvement and Hunt's 54.6 per cent total improvement endows the present writer's own small group with some degree of respectability. The similarity between the two groups now under consideration is so close as to be an actual source of embarrassment to the writer. It is precisely in patients of this sort, that is, patients who under ordinary circumstances have a fair prognosis, that one is prone to form unjustified personal impressions as to the value of insulin therapy. As an operator of an insulin clinic it is both disappointing and enlightening to know that one's personal impressions are not supported by one's own statistical analysis. One further important finding in Table 2 is the very close percentage correlation between each of the corresponding sub-groups. The shift to the improved group in the control cases, which was seen in Table 1 is now absent. This suggests that the absolute chronicity of the larger group which made up Table 1 may have been responsible for the shift.

SUMMARY AND CONCLUSIONS

1. One hundred and sixty-five schizophrenics who received insulin treatment were studied. As time passed the initial total improvement rate of 62.4 per cent leveled off to 40 per cent.

2. This corresponds very closely to the results achieved in a most unfavorable "control" group.

3. There is further correspondence between the results achieved in both of these groups and in some large non-insulin-treated groups abstracted from the literature.

4. Some criticisms of the method of recording results are offered and brief case histories are included to explain the criticisms.

5. Insulin-treated patients whose psychoses were of less than six months duration are compared to a non-insulin-treated group. The two groups show a very marked resemblance to each other. Furthermore, both groups resemble larger groups abstracted from the literature.

6. The value of insulin treatment at the Rochester State Hospital has not been proven. On the contrary, considerable doubt is raised as to its value.

Rochester State Hospital
Rochester, N. Y.

REFERENCES

1. Cheney, Clarence O. and Drewry, P. H.: Results of non-specific treatment in dementia præcox. Am. J. Psychiat., 95:203-217, July, 1938.
2. Hunt, R. C., Feldman, H. and Fiero, R. P.: "Spontaneous" remissions in dementia præcox. PSYCHIAT. QUART., 12:414-425, July, 1938.
3. Malamud, Wm. and Render, Norman: Course and prognosis in schizophrenia. Am. J. Psychiat., 95:1039-1055, March, 1939.
4. Rosenberg, E. F. and Moersch, F. P.: The insulin-hypoglycemia treatment of schizophrenia. Results in treated and untreated cases: Proc. Staff Meet. Mayo Clin., 13:113-116, February, 1938.
5. Halpern, Fanny G.: Insulin shock treatment of schizophrenia. Am. J. Psychiat., 96:1153-1165, March, 1940.
6. Ross, J. R. and Malzberg, B.: A review of the results of the pharmacological shock therapy and the metrazol convulsive therapy in New York State: Am. J. Psychiat., 96:297-316, September, 1939.

THE RETINA AND OCULAR TENSION DURING PROLONGED INSULIN COMA; WITH AUTOPSY EYE-FINDINGS*

BY ALEXANDER GRALNICK, M. D.

In a previous paper,[1] the writer reported the retinal findings in a series of 70 cases of functional psychoses that had received insulin shock-therapy. In it, there was a complete review of the literature dealing with the fundi in untreated mental patients, and with the eye grounds and intraocular tension of treated patients. This paper should be referred to. Since it was written, Powell, et al[2] have made observations of the eyes during insulin coma. They conclude that: (a) The fundus and optic disc become pinker, the veins becoming engorged and the arteries relaxed; (b) the vitreous becomes hazy; (c) the intraocular tension increases but may subside during the coma; and (d) all these changes disappear shortly after the administration of glucose. Unfortunately, they did not make blood-sugar determinations; and they studied only two cases during one period of hypoglycemia.

Inasmuch as the retina is derived from, and is histologically similar to the brain, the writer's first study was made to see what changes occurred in it. Pathological changes occur in the brains of experimental animals suffering hypoglycemic convulsions or comas of several hours duration. Short comas which are terminated by the intravenous injection of glucose do not result in brain alteration. In humans, the bulk of work has been on brains of patients who have died after comas of many hours or days. These brains show extensive degenerative and vascular changes.[3]

The retina is similar to the brain in still other ways. According to Himwich, et al,[4] Lennox,[5] and Wortis and Goldfarb[6] the brain consumes glucose almost exclusively. It has a respiratory quotient of one. In ordinary insulin coma and in irreversible coma with hyperglycemia the brain's metabolic rate is definitely decreased.[4-b] Greig, et al,[7] report that the retina most nearly resembles the brain in its consumption of glucose, and Dickens and Simer[8] say the retina's respiratory quotient is one. In fact, the latter assert that

*Published simultaneously by arrangement with the American Journal of Ophthalmology, October, 1941.

"the retina occupies a quite exceptional position among all animal tissues since it has the highest respiration and highest anaerobic glycolysis of any tissue yet studied."

Adler[9, 10] says that the retina and brain have the highest rate of glycolysis. In an experimental study on cats, he found the average blood sugar 135 mgs. per cent and the sugar content of the aqueous and vitreous 113 mgs. and 64 mgs. per cent respectively. He reports that the retina's glycolytic activity is almost double that of any other ocular tissue. In addition, he says the retina surrounds two-thirds of the vitreous, so that sugar getting to the vitreous from the choroidal or retinal circulation must first be exposed to its glycolytic activity. Consequently, the sugar content of the vitreous is the lowest of any ocular tissue. Furthermore, the sugar concentration is lowest near the retina and higher anteriorly toward the iris. Lindeman[11] says the retina is one of the most actively respiring tissues. In frogs, its metabolism (oxygen consumption) compares quite favorably with the brain's.

The writer's first study showed there were no retinal changes in patients sustaining routine insulin-coma. This report is on the eyes of a woman who died after 10 days of irreversible coma. Study of this case had a five-fold purpose, namely, comparison of (a) the fundus with those of patients who survived treatment; (b) the ophthalmoscopic findings with the microscopic; (c) the histopathology of the retina with that of the brain; (d) the intraocular tension with those reported for routine comas, and (e) correlation of the intraocular tension with the blood and spinal pressures.

Anamnesis and Clinical History

The patient was a 40-year-old, well-nourished, Irish female weighing 139 pounds, who was admitted to Central Islip State Hospital June 12, 1939. After her graduation from public school she had stayed at home because "she was always a home girl who had few friends." Her marked introversion became worse. During the seven years before her admission, she barely talked to members of her family, and she became progressively deafer.

The family, however, found her easy to manage until two weeks before her admission, when she began to have auditory hallucinations. She thought people talked about her, and harbored perse-

cutory delusions. She became apprehensive and agitated, trying to leave home during the night. Hospitalization became imperative.

On admission, physical examination was essentially negative. Her blood pressure was 140/100. Both ear drums were thickened and retracted, but the type of deafness could not be determined because of her uncooperative nature. The blood Wassermann was negative.

Preceding and throughout her insulin treatment the patient's behavior remained about the same. She was inaccessible, tense and perplexed. Frequently she appeared depressed, agitated, and fearful. She expressed numerous paranoid ideas, and admitted auditory hallucinations of a threatening character. No visual hallucinations were reported. Sometimes, she was resistive and uncooperative. Mainly, the woman was seclusive and idle on the ward. She was classified as dementia præcox, paranoid.

On January 12, 1940, therapy was begun, with 15 units of insulin given at 7 a. m. As the treatments continued, she usually went into coma at the end of the third hour or during the fourth hour after injection, and was allowed to stay in coma for periods ranging from 20 minutes to two hours and 10 minutes. Most of the comas lasted well over an hour and were interrupted by gavage with sucrose solution. Sometimes, this had to be implemented with glucose intravenously. One hour and 15 minutes after receiving 50 units as her twenty-fifth treatment a generalized urticaria developed. Sucrose was given, and the rash disappeared two and one-half hours later. After this, coma could be produced only by increasing the dosage of insulin, so that by March 1 she was getting 125 units. In all, she received 2,335 units of insulin in 37 separate treatments given five times a week. There were no convulsions, but 26 comas.

At 7:19 a. m. March 7, the patient received 125 units of insulin intramuscularly. At 8:15, her skin was cold and clammy, and she became restless by 8:30. She had twitchings of her head and extremities at 8:40 and perspired freely by 9:00. Her pulse rose to 120 at 9:45; and the patient was reported in coma at 10:00—in the middle of the third hour after injection. She was tube-fed 140 g. of sucrose at 11:05, and received 100 cc. of 33 per cent glucose intravenously at noon.

Respirations became rapid and irregular, and the blood pressure dropped to 95/80. The patient was placed in shock-position, and one ampule of coramine was given intramuscularly. Thereafter, she received intravenously 100 cc. of 33 per cent glucose at 1:00 p. m., and 100 cc. of 50 per cent glucose at 2:45 and at 7:00 p. m. In addition, she was tube-fed large quantities of orange juice, but aspirations showed a large gastric residue. The Babinski sign was positive bilaterally, and stretching movements and twitchings of the face and extremities were prominent. Decerebrate rigidity was noted frequently.

The patient had suffered a precipitate coma. By the next day, it was definitely irreversible. (Her coma had lasted two hours before she first received intravenous glucose.) She had a high temperature (103.6°), rapid pulse (118) and respirations (30), and in addition to muscular twitchings, she vomited some tube feedings. Wortis and Lambert[12] report such findings as typical of irreversible coma.

The woman remained in coma to the time of her death, with no sign that she would rally. Nutrition was maintained by frequent clyses and infusions of 5 per cent glucose in saline, as well as by gavages of orange juice and egg-nog which she retained well. Up to the day before she died, heart sounds remained good, the pulse rate fluctuated between 80 and 160, reaching the latter figure toward the end, and the blood pressure ranged from 115/75 to 150/110. Her temperature ranged between 99.4° and 104°, mounting to 107° the last day. As a rule, respirations were regular, and they varied from 24 to 40. She perspired very freely and eliminated well, though she was incontinent. The Babinski sign continued to be positive bilaterally. Results of blood chemistries and spinal fluid pressures are shown on the chart. (Figure 1.)

About 18 hours before death, the patient went into a rapid decline. Her pulse increased in rate, becoming weak and thready, and respirations became rapid and labored. The blood pressure dropped precipitately, so that six hours before death it could not be obtained. She perspired profusely and had frequent, watery, yellow stools. All extremities became cold and cyanotic, and the temperature gradually rose to 107°. Breath sounds were clear in both lungs. The patient died 10 days and six hours after coma began.

Technique of Study

Throughout her coma the patient's pupils were maintained in a semi-dilated condition with 2 per cent homatropine to make study of the fundus easier. Ophthalmoscopic examination was done first, with careful search made primarily for retinal hemorrhage and optic disc injection or edema. The woman was then placed in the supine position and the blood pressure read. Following this, three ocular tension readings were made with the Schiötz tonometer holding a 7.5 gm. weight. The average was taken as the tension for the moment. This procedure was followed morning, early and late afternoon, and sometimes in the evening. During the night the patient was not disturbed. Spinal pressure readings were made in the morning shortly after the ocular tension was taken, and the blood sugar was determined at the same time.

Findings

The Fundus: Up to the time of death ophthalmoscopic findings were negative except for moderately dilated veins the last day. The retinae showed no hemorrhages, exudates or pigmentary changes, and the discs remained normal in outline and color. The vessels maintained their normal ratio in caliber.

Intraocular Tension: Reference to the graph (Figure 1) will show that the intraocular tension remained low throughout the coma. It ranged from 21.4 to 13.0 in the left eye, and from 19.0 to 11.4 mm., mercury pressure, in the right eye, and declined to 9 mm., mercury pressure, one hour and 15 minutes before death. The tension of the eyes paralleled each other closely and remained more or less on an even plane until four days before death, when a gradual decline set in.

There is no relation between the blood pressure and intraocular tension, the latter remaining low despite a consistently high diastolic pressure. About 18 hours before death, vascular collapse set in. It is indicated on the graph by the acute drop in the blood pressure curve after the twenty-sixth reading. At 9:15 a. m., six and one-half hours before death, the blood pressure could no longer be obtained, but the intraocular tension at this time was 10 mm.; and one hour and 15 minutes before death, it was 9 mm., mercury

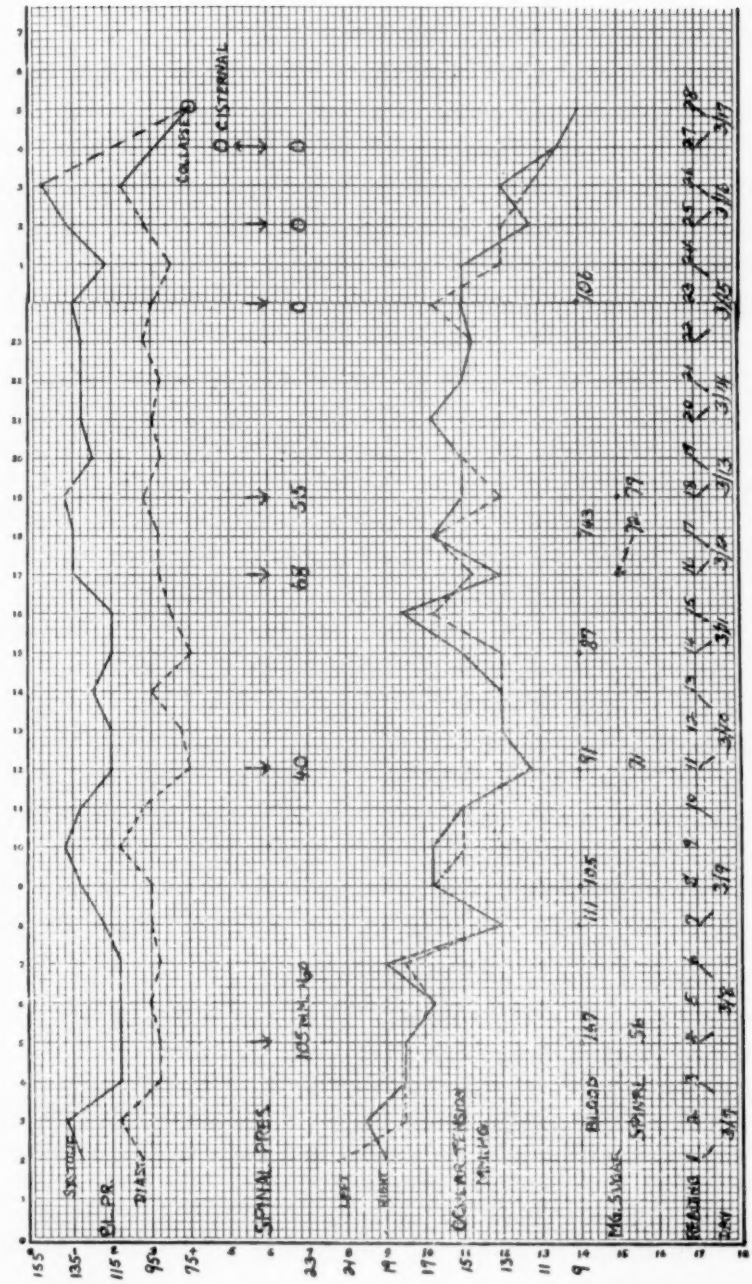


Fig. 1. Graph showing relation of ocular tension to blood- and spinal-pressures and sugar-levels.

pressure. At the moment of death, intraocular tension could not be measured because the eyes were too soft.

The graph shows that there was no direct correlation between the spinal pressure and ocular tension. However, in the four instances that a spinal pressure could be obtained the ocular tension fluctuated with it. Thus, when the spinal pressure was 105 mm., water pressure, the ocular tension was 18 mm., mercury pressure; when spinal pressure was 40 mm., the eye tension was 11.4 mm.; when spinal pressure was 68 mm., the tension of the left eye was 14.6 mm.; and when the spinal pressure was 55 mm., left eye tension was 13 mm. The zeros on the graph indicate unsuccessful attempts to register the spinal pressure. In these attempts, a few drops of fluid could be obtained, but none would rise in the manometer. A cisternal puncture was attempted the morning of death, but no fluid was obtained. It should be noted that during the three days before death, the ocular tension slowly declined; and, at autopsy, little spinal fluid was found in the ventricles.

The intraocular tension remained uniformly low, despite normal or elevated sugar levels in both the blood and spinal fluid.

One other finding is worthy of mention. Forty-five minutes before death, the eyes showed rapid, synchronous, oscillatory movements in the horizontal plane. These lasted for about one-half hour. The oscillation was no more than 2 mm. in either direction from the mid-point at which the eyeballs seemed to be fixed. This movement sometimes stopped momentarily on expiration, but did so with no regularity.

Autopsy Report

The postmortem examination: This was done by Dr. Reider Trygstad of Central Islip State Hospital. It follows:

The autopsy is begun 45 minutes after death. General external examination reveals a well-developed white female. The brain weighs 1,370 grams. It is edematous, and the sulci are shallow and narrow. The meninges are transparent and have markedly congested vessels. The basal arteries are decreased in caliber. There are no areas of softening or atrophy. The ventricles are smaller than normal and contain a dinimished amount of fluid.

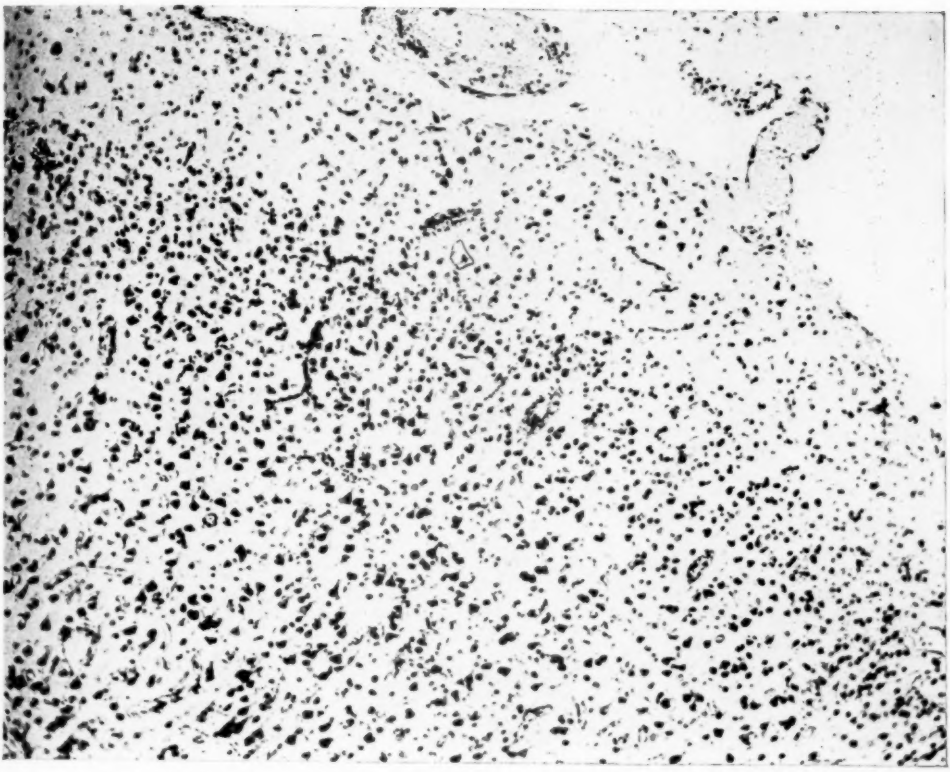


Fig. 2. High power magnification of the cortex to illustrate increased vascularization of various cortical layers.

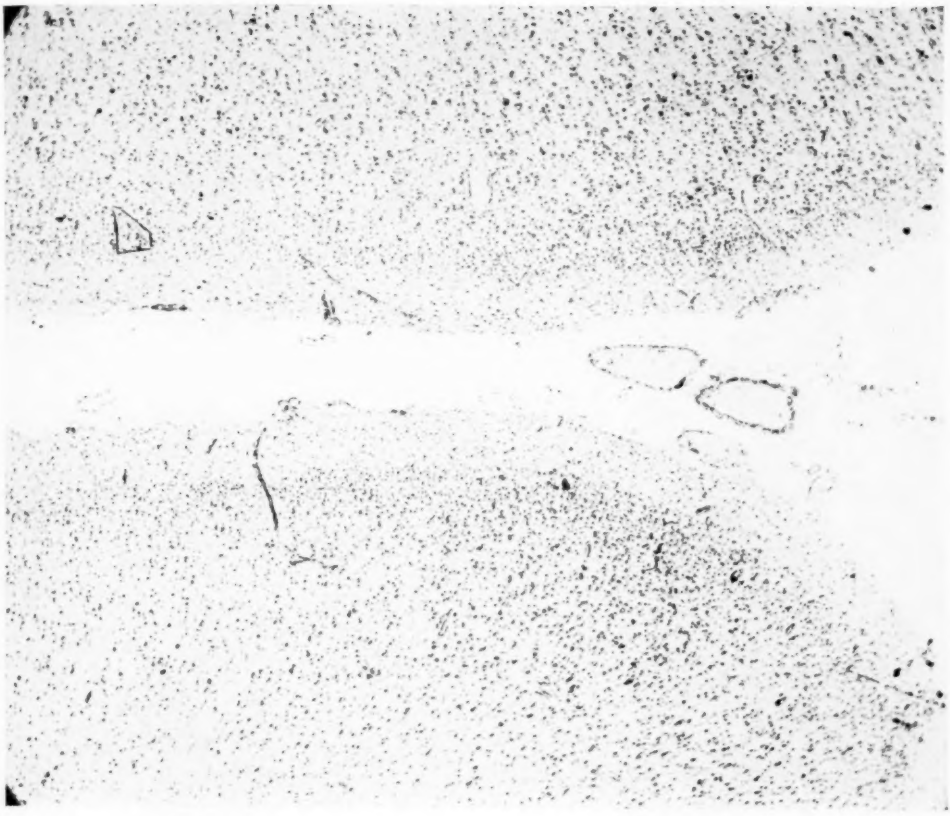


Fig. 3. Low power magnification of cortex to show the rarefaction of nerve cells and consequent disturbed lamination in addition to the increased vascularity and endarteritic changes.

The chest cavity is negative and the lungs are congested at their bases. The heart weighs 340 grams and is uniformly dark red. The aortic valve measures 5.5 cm. and the pulmonary 6.5 cm. in circumference. The abdomen shows the serous linings to be smooth and glistening. The adrenals are of average size, and show congestion of the medullae. The remaining abdominal organs are essentially negative. The eyes are placed in Zenker's solution immediately after removal.

Microscopic Examination of the Brain: Nissl Stain

This was done by Dr. Armando Ferraro of the New York State Psychiatric Institute and Hospital and Dr. Max Gold of Central Islip State Hospital through collaboration in their respective pathology laboratories. It follows:

In the meninges, there is a considerable amount of congestion which extends within the cortex and white substance. The veins are generally the ones involved, the dilatation being very pronounced. In the external layers of the cortex, the blood vessels are often seen entering from the meninges and disclosing thickening of their walls. Some of the blood vessels can be followed up to the second and, occasionally, to the third layer. (Figure 2.) In addition to these individual long blood vessels, one finds, particularly in the lamina molecularis, numerous small blood vessels, disclosing also thickening of their walls. The thickening of the walls is at the expense of the intima in the very small blood vessels and of the outer layers and the intima in the middle-sized ones. The endarteritic process is found also here and there in the white substance. As far as proliferation of blood vessels is concerned, one notices that there is a certain amount of newly formed blood vessels, but this neoformation is definitely less pronounced than the ones noticed in other cases of insulin encephalopathy.

The nerve cells in the various layers of the cortex are still present in large number sufficient to preserve the normal cytoarchitecture. There is, however, a certain rarefaction of nerve cells distributed all over the various cortical layers. (Figure 3.) Here and there, cortical areas are found in which the cytoarchitecture is somewhat disturbed; and there are other areas, much less numerous, in which the disturbed lamination is quite pronounced. In the cortex, generally speaking, one encounters a more pronounced pathological process in the lamina molecularis where the vascular component of the pathological process is more prominent. In the remaining layers of the cortex, nerve cells are seen undergoing various degenerative changes, mostly chromatolysis. Degenerative process in the nerve cells leads in certain areas to the severe type of degeneration, and remnants of nerve cells as well as shadow cells are seen here and there.

In the white matter one finds with various degrees of intensity a glia proliferation, as expressed principally in increase of glia nuclei. Especially surrounding the blood vessels and along the blood vessel walls, one sees collection and rows of glia nuclei, presumably nuclei of oligodendroglia cells. Fat products of degeneration are found in the various cortical layers in correspondence particularly to the areas where the nerve cell degeneration is most pronounced. Fat degenerative products are found in the perivascular spaces and within the nerve cells.

Concerning the distribution of the pathological findings, one finds that the frontal, temporal and occipital areas are more severely damaged than the remaining parts of the brain. In the temporal lobe, it is particularly Ammon's horn that shows more pronounced vascular and cellular changes. (Figure 4.) In the Sommer sector of Ammon's horn, one finds a considerable destruction of nerve cells which at low power gives the impression of discontinuity of the lamina pyramidalis. Fat products of degeneration are more abundant in Ammon's horn, particularly within the nerve cell elements. In the cerebellum, vascular changes in the direction of endarteritis are more pronounced in the lamina molecularis. Homogenization of the Purkinje cells is a common finding and in the white substance, glia proliferation of the cerebellum nuclei is quite pronounced. No appreciable clumping of granular cells is noted.

Comment: Altogether, we are dealing in this case with a diffuse encephalopathy, non-specific, characterized by two main histopathologic features—involvement of the vascular system and nerve cells. The pathological findings of the vascular system are along the lines already described in other cases of insulin encephalopathy, that is, hypertrophy and hyperplasia of blood vessels. The endarteritic process is, however, more pronounced here than the proliferative one. Both vascular hypertrophy and hyperplasia are present in a moderate degree.

Involvement of the nerve cells consisting mainly of degenerative changes leads to a certain amount of destruction of the elements, but not in any pronounced degree. Thus the cytoarchitecture is affected only here and there, particularly in the frontal, temporal and occipital areas.

Histopathology of the Eye: The eye was examined by Dr. L. V. Sallman, of the ophthalmology department of the Columbia University College of Physicians and Surgeons. His report follows:

Cornea: Epithelium cover absent in parts and in others reduced to two or three layers, with destruction of normal arrangement. Corneal parenchyma, elastic membranes and endothelium normal. Pigment deposits at the posterior surface.

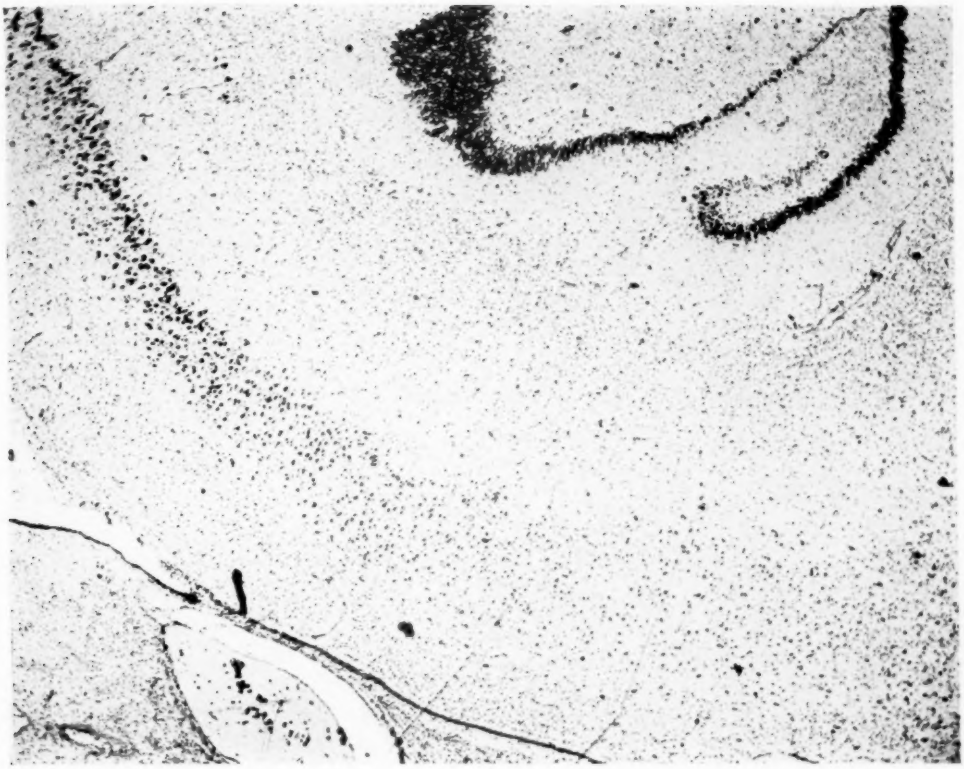


Fig. 4. This illustrates the disturbance of nerve cells in the lamina pyramidalis of Ammon's horn.

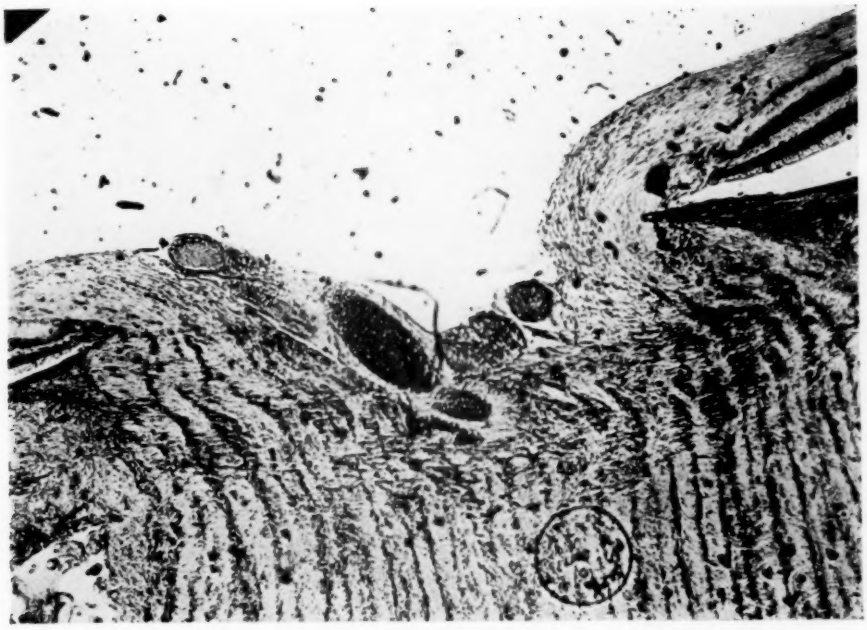


Fig. 5. Low power magnification to show normal optic cup with engorgement of the vein.

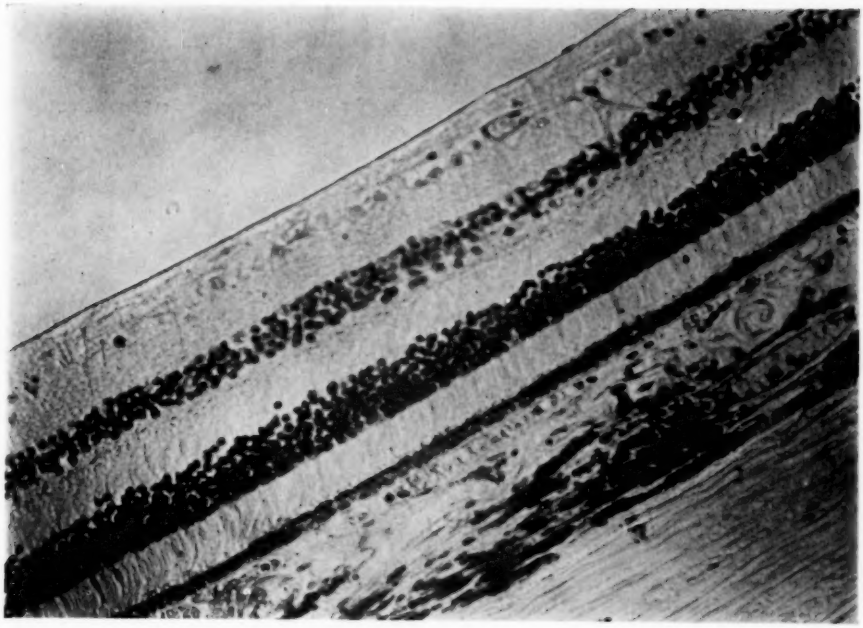


Fig. 6. Moderately high-power magnification of retina showing normal architecture.

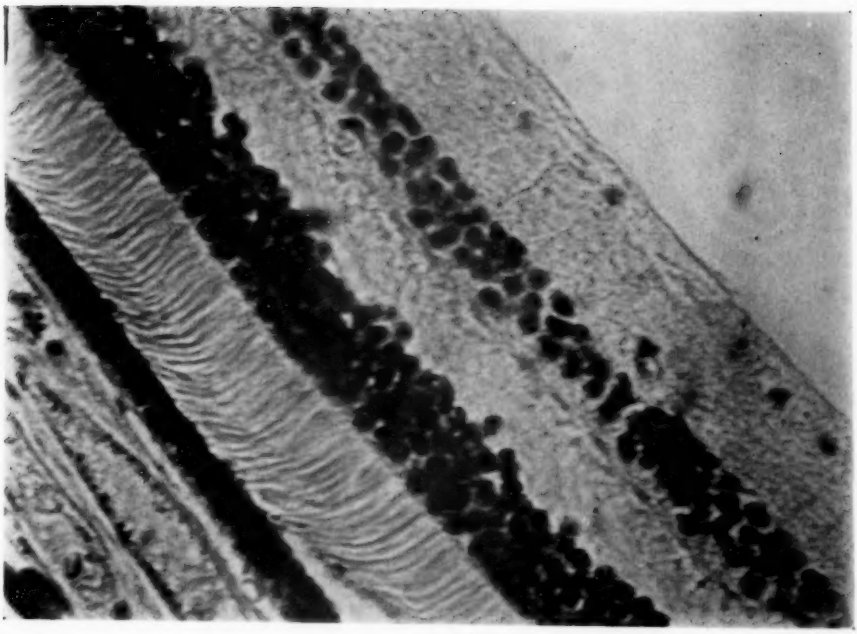


Fig. 7. High power magnification of same section of retina as in Fig. 6 showing cellular detail.

Anterior Chamber: Normal in depth and content. Filter pectine shows a great number of pigment deposits. Angle of anterior chamber is free.

Lens: Capsule and epithelium normal. Nucleus removed in imbedding.

Iris and Ciliary Body: Normal.

Choroid: Normal *in toto.*

Retina (Figures 5, 6 and 7): Artificially detached. In a good state of preservation. The layers are normal in thickness. There are no signs of pathological changes in the nerve fiber layer. Ganglion cells (hematoxylin and eosin, Mallory and different forms of Nissl stain) are mainly normal in number and structure. The nuclei are well preserved with the exception of a few hyperchromatic nuclei lying in an acidophile mass. Nissl bodies are on the whole unstained (Zenker's fixative). A very small number of them can be demonstrated lying normally close to the cell periphery in a semi-circle. There is no vacuolization of the cytoplasm. The inner and outer granular layers are also well preserved but a few cells show pyknotic nuclei with chromatin clumping. Rods and cones seem to be normal insofar as can be determined in celloidin sections.

Optic Nerve: Does not show any lesion in hematoxylin and Weigert sections. Marchi's stain was not applicable because of the fixative.

Diagnosis and Discussion: Abrasion of cornea with regenerative changes of epithelium. Scattering of pigment in anterior chamber. Artificial retinal detachment.

The changes of the ganglion cells and the granular layers cannot be evaluated on account of the fixative. The pyknotic changes in the granular layers are not extensive enough to be considered pathological, but the possibility of a slight degree of degeneration cannot be ruled out.

Summary and Discussion

The literature contains no report on the eye during irreversible insulin coma. Although many brains have been studied, the retina —which is a similar structure—has not been. This paper reports a study of the eye during 10 days of insulin coma. It describes the retina during life and its histopathology in relation to that of the brain after death. It also shows the correlation between the intraocular tension and the blood pressure and spinal pressures on the one hand, and the correlation between the intraocular tension and the blood sugar and spinal sugar levels on the other.

The brain pathology will not be discussed here because it has been covered in comprehensive studies already in the literature.[3] It need only be mentioned that in this case the brain changes are typical and extensive.

The retina bears several striking similarities to the brain. Histologically it has like cellular structure, and embryologically it is a derivative of the brain. In addition, it too has a respiratory quotient of one, indicating that its chief nourishment is glucose. One could logically assume that because of these similarities the pathology in the retina would resemble that in the brain. This, however, does not seem to be the case.

The retinal changes are slight. Those reported here may be due to the coma, but it is just as likely that they are postmortem in nature. Certainly, with the extensiveness of the brain pathology, the retina might be expected to show marked changes. Instead it "is in a good state of preservation," and the ganglion cells, so markedly altered in the brain, are "mainly normal with the exception of a few hyperchromatic nuclei."

Although more similar cases must be studied, it is allowable to speculate on the physiological mechanisms proposed to explain the brain pathology,[13] and see if the present findings support them. The chief supposition is that insulin hypoglycemia causes an intracellular anoxemia which eventually destroys the brain-cells. Wortis[14] and Himwich[15] say the brain's diminished metabolism produces the pathology, for in reality there is no anoxemia. The retina's metabolic rate compares favorably to the brain's. The writer does not know, however, the way the retina's metabolic rate is affected in irreversible comas of this type. Nonetheless, from the evidence, one would expect a decrease in it, too, with resulting damage. It is curious that this does not occur.

A second theory, that vascular disturbance produces the brain damage, is not well supported by the evidence in this case. The retina shows no focal areas of necrosis, and the vessels are all normal. Even the layer of rods and cones, which has no direct blood supply of its own but gains nourishment from the contiguous choriocapillaris, does not suffer. Perhaps the retina is saved because its blood supply is richer than the brain's.

If the third concept, that insulin acts as a toxin directly on the brain-cells is correct, why then does the retina escape? It is very sensitive to other toxins, and should react to insulin if the latter is a damaging agent and is given in large doses.

Himwich[15b] states that the clinical changes occurring in hypoglycemia show that the newer portions of the brain are first affected. Later the functioning of phylogenetically older layers is affected. Heymans[16] says that acute anoxia shows the cerebral cortex most vulnerable, and the upper portion of the brain-stem and medulla less vulnerable in that order. With the aid of electrograms, Hoagland[17] shows that during hypoglycemia, the activity of the cerebral cortex is depressed before that of the hypothalamus.

From the foregoing, Himwich[15b] supposes that the older portions of the brain are less sensitive. They have smaller energy requirements and are therefore more resistant to hypoglycemia than the younger parts, with their higher metabolic rate. Thus, when sugar is given, the older parts are the first to recover. All of this may account for the phylogenetically-older retina's apparent resistance to damage during the coma. In addition the blood sugar level of this patient could have been high enough to spare the retina but not the brain. The disconcerting feature, however, is that studies [7, 8, 9, 11] show the retina's metabolic rate to be very high. It should thus be responsive to hypoglycemic-changes despite its older phylogenetic age.

The intraocular tension in the case presented was uniformly low, declining to very low levels in the last days of life. This finding agrees with those of many who have made a similar observation during routine insulin comas.[18] The ocular tension showed no correlation with the blood pressure or sugar levels of the blood and spinal fluid, but did fluctuate, somewhat inexactly, with the spinal pressure.

The spinal fluid pressure was uniformly low. This fact, coupled with the diminished amount of fluid in the ventricles, and the failure to obtain a spinal pressure during the last three days despite cerebral edema may account for the lowered intraocular tension. Obviously there was some defect in the system that forms spinal fluid and maintains its pressure. Perhaps this was in the choroid plexus, for the blood pressure stayed at a good level.

Ophthalmoscopic examination of the retina was normal during life, and confirmed by the microscope. This evidence supports the writer's previous finding[1] that insulin coma, routinely given, does not affect the retina.

Conclusions

In prolonged insulin coma of 10 days duration:

1. The retina appears to escape damage, although the brain sustains extensive injury.
2. The ophthalmoscope reveals no gross pathology. This finding supports one reported in a previous paper—that the fundi are negative in functional psychoses surviving routine insulin shock therapy.
3. The intraocular tension remains uniformly low, and fluctuates inexactly with the spinal pressure.
4. The intraocular tension has no relation to the blood pressure, or blood and spinal sugar levels.
5. The finding of lowered intraocular tension corresponds to that of most investigators who report the same in routine insulin comas of short duration.

Acknowledgment

The writer wishes to express his thanks to Dr. Marcus Schatner of Central Islip State Hospital for his cooperation, and to Dr. E. Burchell of the New York Eye and Ear Hospital for preparation of some of the eye-slides studied.

Central Islip State Hospital
Central Islip, N. Y.

REFERENCES

1. Gralnick, A.: The eye grounds in functional psychoses given insulin shock therapy: Psychiat. Quart., 15:1, 74-85, January, 1941. Am. J. Ophth., 24:1, January, 1941.
2. Powell, L., Hyde, M., and Russell, J.: Observations on the eyes during insulin shock treatment. J. Kans. Med. Soc., 41:97, March 1940.
3. Papers reporting brain pathology:
   a) Weil, Leibert, and Heilbrun: Histopathologic changes in the brain in experimental hyperinsulinism. Arch. Neur. and Psychiat., 39:467-481, March, 1938.
   b) Reed and Dancey: Insulin therapy of dementia præcox. Internat. Clin., 2:101-125, June, 1938.

c) Accornero, F.: Experimental histopathologic researches on shock. Am. J. Psychiat. (suppl), 94:130-33, May, 1938.
d) Ferraro, A., and Jervis, G.: Brain pathology in four cases of schizophrenia treated with insulin. PSYCHIAT. QUART., 13:22, April, 1939.
e) Malamud, N., and Grosh, L.: Hyperinsulinism and cerebral changes. Arch. Int. Med., 61:579-599, April, 1938.
f) Roizin, L.: Histologic alterations of the central nervous system in animals subjected to insulin shocks. Riv. di Patol, Nerv e Mentale, 53:491, May, 1939.

4. a) Himwich, H., Bowman, K., Wortis, J., and Fazekas, J.: Biochemical changes occurring in the cerebral blood during insulin treatment. J. Nerv. Ment. Dis., 89:273-293, March, 1939.
b) Himwich, H., et al: Metabolism of the brain during insulin and metrazol treatments. J. A. M. A., 112:1572-73, April 22, 1939.

5. Lennox, W.: The cerebral circulation: Respiratory quotient of brain and extremities in man. Arch. Neur. and Psychiat., 26:719, October, 1931.

6. Wortis, J., and Goldfarb, W.: Substrates for brain metabolism during insulin shock. Science, 91:270-71, March 15, 1940.
Wortis, J., and Goldfarb, W.: Availability of glucose for human brain oxidations. Proc. Soc. Exptal. Biol. Med., 44:382-85, 1940.

7. Grieg, F., Munro, M., and Elliott, K.: Metabolism of ox retina and chick embryo. Biochem. J., 33:443, 1939.

8. Dickens, F., and Simer, F.: Metabolism of normal and tumor tissue. Biochem. J., 24:1301, 1930.

9. Adler, F.: The metabolism of the retina. Arch. Ophth., 6:901, 1931.

10. Adler, F.: The sugar content of the ocular fluids. Tr. Am. Ophth. Soc., 28:307, 1930.

11. Lindeman, V.: The respiratory metabolism of the frog retina. Physiol. Zool., 13:411, October, 1940.

12. Wortis, J., and Lambert, R.: Irreversible or hyperglycemic insulin coma. Am. J. Psychiat., 96:335-45, September, 1939.

13. Papers reporting on physiological mechanisms:
a) See 3a.
b) Dameshek, W.: Mechanisms of the neurologic symptoms in insulin hypoglycemia. Arch. Neur. and Psychiat., 33:18, January, 1935.
c) Gellhorn, E.: Action of insulin hypoglycemia on the central nervous system. J. A. M. A., 110:1433-34, April 30, 1938.
d) Grayzel, D.: Changes in the central nervous system due to insulin convulsions. Arch. Int. Med., 54:694, November, 1934.

14. Wortis, S.: Effects of insulin on brain metabolism. N. Y. S. J. Med., 38:1015-21, July, 15, 1938.

15. a) Himwich, H., Bowman, K., and Fazekas, J.: Prolonged coma and brain metabolism. Arch. Neur. and Psychiat., 44:1098, November, 1940.
b) Himwich, H., et al: Mechanism of the symptoms of insulin hypoglycemia. Am. J. Psychiat., 96:371, 1939.

16. Heymans, C., et al: Survival and revival of nerve centers following acute anemia. Arch. Neur. and Psychiat., 38:304, August, 1937.

17. Hoagland, H., Himwich, H., et al: Effects of hypoglycemia on electrical activity. J. Neurophysiol., 2:276, 1939.

18. Papers reporting decreased intraocular tension:
a) Ciotola, G.: Behavior of ocular tonus in insulin coma. Boll. d'Ocul., 17:738-54, September, 1938.
b) Wolf and De Jongh: Insulin and reduction of ocular tension. Klin. Wochenschr., 41:344, 1925.
c) Mamola, P.: Effects of insulin on ocular tension. Riv. san Sicil., 21:431-37, March 15, 1933.
d) Weichmann, E. Difficulties in diagnosis of insulin coma. J. A. M. A., 92:1495-96, May 4, 1929.
e) Vestergaard, J.: Insulin and ocular tension. Acta. Ophth., 7:273-300, 1929.
f) Raiha, C., and Gronlund, L.: Effects of insulin on intraocular tension. Skandinav. Arch. f. Phys., 62:70, July, 1931.

SOME OBSERVATIONS ON CARDIOVASCULAR CHANGES IN SHOCK THERAPY

BY HERVEY CLECKLEY, M. D., AND DU BOSE EGLESTON, JR., M. D.*

Early reports, discussing shock therapy with metrazol[1] and with insulin,[2] mention, as a contraindication in these methods, the presence of serious cardiovascular disorder. In the course of insulin therapy, arrhythmias "ranging from extrasystoles to auricular fibrillation" have been reported.[3] After the seizures induced by metrazol, cyanosis is the rule.[4] Since this cyanosis sometimes is sufficiently severe to be alarming to the observer, it is not surprising that the possibility of cardiovascular damage from metrazol should, from the advent of this therapy, have received considerable attention.

Meduna[5] reported that cardiovascular studies on a series of patients who had completed metrazol treatment revealed no significant changes. The report by Hayman and Brody[6] of a death following convulsive treatment in a patient found at autopsy to have had endocarditis emphasized the probable stress thrown on the heart by metrazol in convulsive doses. Geraudel[7] reported transient arrhythmias and a number of brief minor electrocardiographic changes after convulsions. He also noted in two cases inversion of the T wave in leads three and four which lasted as long as three days. This last finding was considered to be an indication to discontinue metrazol therapy. Orenstein[8] also mentioned electrocardiographic changes but considered them to be temporary. Schilder[8] reported the interruption of treatment in a patient who showed particularly severe irregularities. Dick and McAdam[9] observed auricular fibrillation which lasted three days, partial heart block of several minutes duration and right axis deviation which was still present three weeks after treatment with metrazol. Pellens[10] reported changes in the P-R segment, depression of the S-T segment, variations in the T wave and various arrhythmias, all observed immediately after convulsive treatment. No evidence was offered to indicate that these changes were lasting. McAdam[11]

*From the departments of neuropsychiatry and medicine, University of Georgia, School of Medicine.
The writers acknowledge their appreciation to Dr. H. T. Harper of the department of cardiology, University of Georgia School of Medicine, for his very valuable cooperation in this study.

subsequently reported further observation of auricular fibrillation after metrazol and also of reduplication in the second mitral sound. These abnormal phenomena disappeared promptly, and no evidence of myocardial damage persisted. Forschbach[12] reported the alarming development of cardiac standstill, which lasted for four minutes, and also of heart block, inversion of the T wave and other electrocardiographic changes. All of these changes were noted shortly after treatment. Strecker and his associates[13] also observed auricular fibrillation, water-hammer pulse, capillary pulsation and other signs of disturbed cardiac function, but found these effects to be transient. Gross and Gross-May[14] noted auricular fibrillation which persisted for four hours, syncope and other signs of temporary cardiac disorder. Rathnell,[15] in studies made a year or more after metrazol treatment, found evidence of sinus tachycardia and changes in the S-T segment and the T wave which were interpreted as probably dependent on the increased cardiac rate. Orenstein[16] noted tachycardia, changes in the T wave and the S-T segment, arrhythmias and other marked but temporary cardiovascular changes. Messinger and Moros[17] observed temporary arrhythmias and increases in pulse rate but found "in no instance . . . the slightest significant change, or evidence of cardiac damage as a result of the treatment." These workers conclude that "metrazol may be employed with a feeling of much greater assurance in borderline cardiac cases than would be the case with insulin . . ." Electrocardiographic study, in many instances, indicated "improved coronary oxygenation associated with the more vigorous heart action." Levine and his coworkers[18] also noted an increase in heart rate, changes in the T waves and other electrocardiographic variations, all of which were transient. Bennett's report[19] of metrazol therapy in affective psychoses, in which convulsions were successfully given to patients as old as 68 years, might be regarded as evidence that this regimen does not always put an extremely severe burden on the cardiovascular system.

In the writers' own series of patients, significant cardiovascular changes were not found clinically, or in the electrocardiogram after treatment, except in a few instances. The following cases, however, impressed the writers as offering points of interest in this discussion.

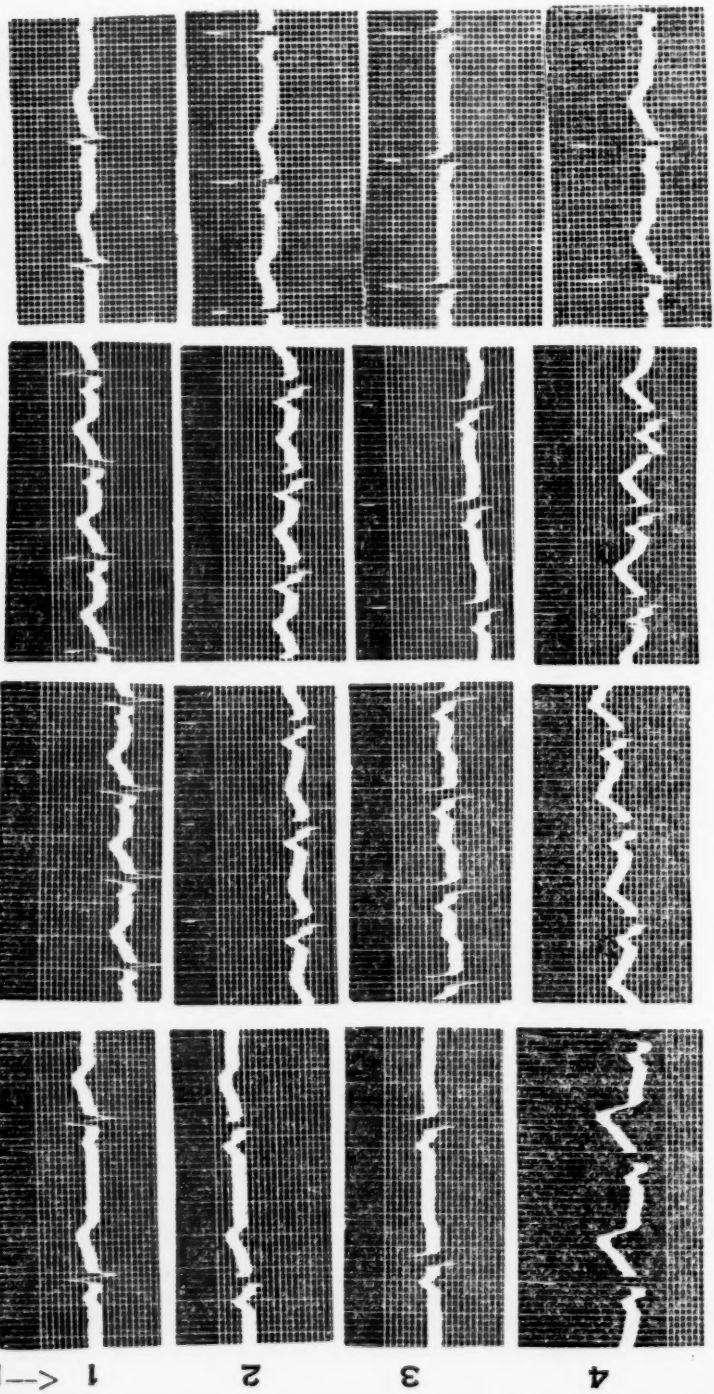


FIGURE 1

Case 1. N. N., colored male, 19 years of age, admitted to the University Hospital, University of Georgia, on August 17, 1939. The personality status was typically that of schizophrenia, hebephrenic type. Physical, neurologic and laboratory examinations revealed nothing significant.

Electrocardiogram, August 17, 1939. (Figure 1A.)
Rate: A-62 V-62
Rhythm: Regular
P. R.: 0.16
QRS: 0.06
Reading: Low T_3
Impression:
Normal sinus mechanism
Physiologic curve

Metrazol therapy was begun on August 19, with 4 cc., which produced a typical convulsion. After having received eight administrations of metrazol which resulted in six uneventful seizures, on August 31, the patient again received metrazol, 6 cc. A typical but unusually severe convulsion occurred consisting of (1) gross unsynchronized jerks, five seconds, (2) tonus with marked opisthotonous, 15 seconds, (3) clonus, 55 seconds. At the end of the convulsion, cyanosis was marked. Respiration was regained with difficulty. For several minutes, periods of apnea (15 to 20 seconds) alternated with strained, grunting, laborious efforts to breathe. The pulse was rhythmical but varied in rate (120-45-100). For two or three minutes more, some respiratory embarrassment continued. Cyanosis, instead of subsiding with a restoration of free respiration as in the writers' previous experience, persisted, the finger nails and mucous membranes showing vividly blue 20 minutes after the seizure. This cyanosis gradually diminished but could be observed for an hour.

These reactions, particularly the persistent cyanosis, were considered a possible contraindication to convulsive therapy and metrazol was discontinued. Five days after the last treatment a second electrocardiogram was made. Meanwhile (on September 2, 3, and 5), insulin had been given, but not in sufficient quantity to produce any apparent effect.

Electrocardiogram, September 5, 1939. (Figure 1B.)
Rate: A-110 V-110
Rhythm: Regular
P. R.: 0.13
QRS: 0.05
Reading: Deep S_1; Blunt T_2; Diphasic T_3; Depressed ST_3

Impression:
S. A.
Tachycardia
Slight right axis deviation
Myocardial damage, slight

Insulin therapy was continued daily. Deep, quiet coma was not obtained, but, with 120 to 160 units, motor hyperactivity, excitement, grimacing and sucking were produced. A marked vocal uproar accompanied these reactions, and persistent stereotypy was noted. For long periods, he yelled time after time, "George Washington are the ruler of Great Britain!" This sentence, unaltered in syllable but marvellously varied in rhythm and accent, he used in replying to all questions. He also gave it spontaneously in whispers, in roars, in lively, scanned beat and in lugubrious monotone. The appearance of ecstasy, indifference, politeness, rage, defiance, intense interest and coyness, in fitful succession, accompanied the words.

On September 12, 1939, 12 days after the last injection of metrazol, a third electrocardiogram was made.

Electrocardiogram, September 12, 1939. (Figure 1 C.)
Rate: A-100 V-100
Rhythm: Regular
P. R.: 0.13
QRS: 0.07
Reading: Slight slurring of QRS waves
Diphasic T_3; Q_3 equals minus 2½ mm.; Q_4 equals minus 2 mm.
Impression: Normal sinus mechanism
Better voltage of T_2 than in the tracing of September 5
Minor changes in QRS complex

Insulin therapy at this time was discontinued and, as the patient had not improved, he was admitted to the state hospital.

On June 26, 1940, almost 10 months after the last treatment with metrazol, a fourth electrocardiogram was obtained.*

Electrocardiogram, June 26, 1940. (Figure 1D.)
Rate: A-70 V-70
Rhythm: Sinus arrhythmia
P. R.: 0.16
QRS: 0.06
Reading: Low QRS_1; Low T_3

*The writers acknowledge their appreciation to Dr. John W. Oden, superintendent of the Milledgeville State Hospital, Milledgeville, Ga., for his kindness and cooperation.

Impression:
Sinus arrhythmia
Physiologic curve

No clinical evidence of impaired cardiovascular function has been noted since the disappearance of the cyanosis an hour after the last treatment with metrazol.

Case 2. L. E., white female, aged 33, admitted to the University Hospital on December 5, 1938. Physical, neurologic and laboratory examinations revealed nothing significant. Her psychiatric picture was that of schizophrenia, catatonic type.

Electrocardiogram, December 12, 1938 (prior to treatment). (Fig. 2A.)
Rate: A-90 V-90
Rhythm: Normal
P. R.: 0.14
QRS: 0.05
Reading: Normal
Impression:
Normal sinus mechanism
Physiologic curve

Treatment with metrazol was begun December 16, with 3 cc. No seizure was obtained until December 20, when, on the sixth injection (7.5 cc.), a typical seizure was obtained. Metrazol was continued through December 28, being given every other day when a seizure was obtained. When a seizure was not obtained, the dose was increased and repeated after one hour. Ten injections of metrazol were given, but only three major seizures were produced. The patient had apparently recovered and was dismissed.

On January 12, 1939, signs of the psychosis had returned; she was readmitted and metrazol therapy resumed. She received 13 injections (6 to 10 cc.) of metrazol and responded with eight major seizures. Her seizures were violent, and respiration was often regained only with much labor, dyspnea and cyanosis persisting for several minutes after the convulsions and the conjunctival vessels remaining markedly engorged. After the 11th injection in this series (seventh seizure) a second electrocardiogram was made.

Electrocardiogram, January 26, 1939. (Not in figure.)
Rate: A-100 V-100
Rhythm: Regular
P. R.: 0.14
QRS: 0.08
Reading: Elevated ST_3

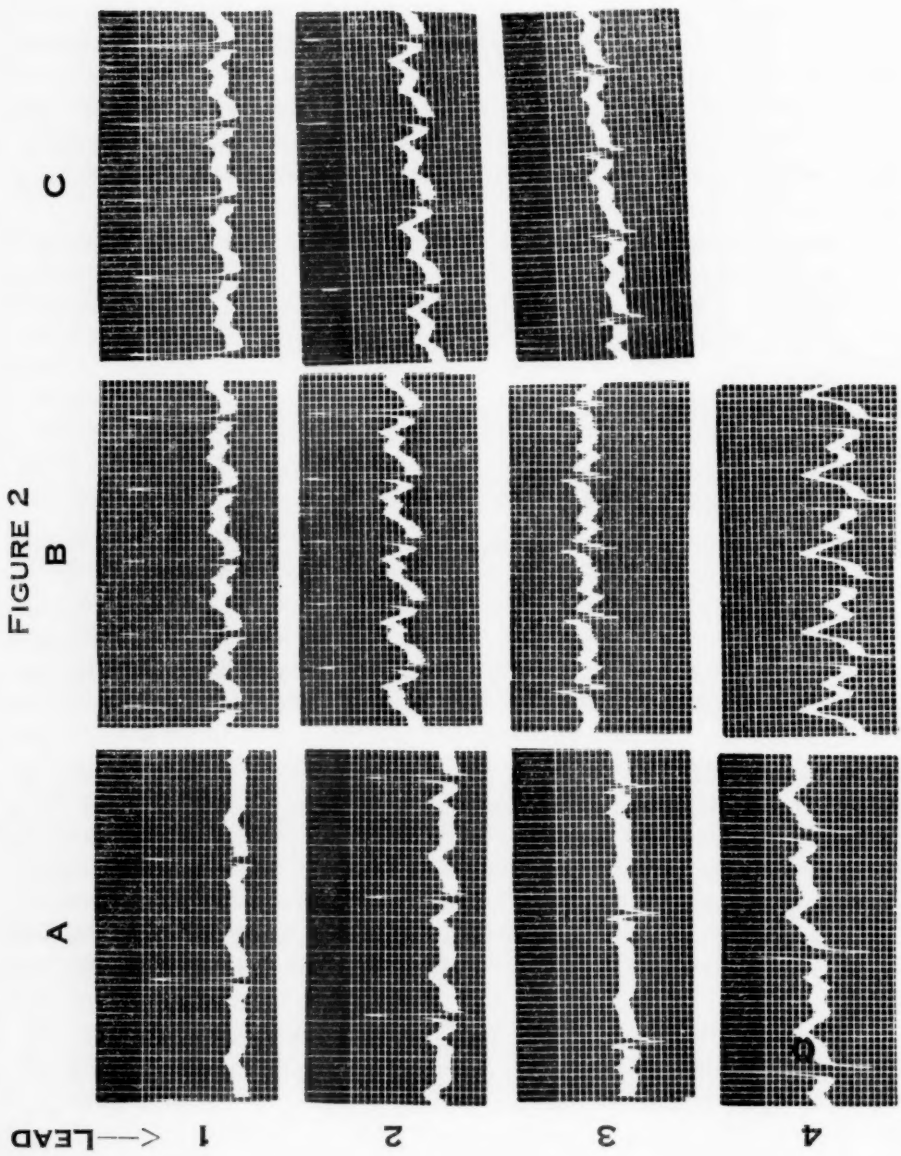


FIGURE 2

Impression:
Normal sinus mechanism
Physiologic curve

Metrazol therapy was continued. All signs of the psychosis subsided and on January 29, 1939, the patient was dismissed, apparently in normal condition.

When seen at the outpatient clinic on February 3, 1939, she was still free from signs of psychosis but complained of dyspnea and of substernal and epigastric pain. Examination revealed moderate dependent edema, tachycardia (140-150) and dyspnea on the slightest exertion.

Electrocardiogram, February 3, 1939. (Figure 2B.)
Rate: A-150 V-150
Rhythm: Regular
P. R.: 0.12
QRS: 0.06
Reading: Depression of S-T segments in Leads 1, 2 and 3
Impression: S. A. Tachycardia
Change in S-T segments compatible with rapid rate

The cardiologist at this time advised observation feeling that myocardial damage might have occurred. A third electrocardiogram was obtained one week later.

Electrocardiogram, February 10, 1939. (Not in figure.)
Rate: A-115 V-115
Rhythm: Regular
P. R.: 0.13
QRS: 0.07
Reading: There are variations in the QRS waves, S_3 is deep
Impression: S. A. Tachycardia. Otherwise normal

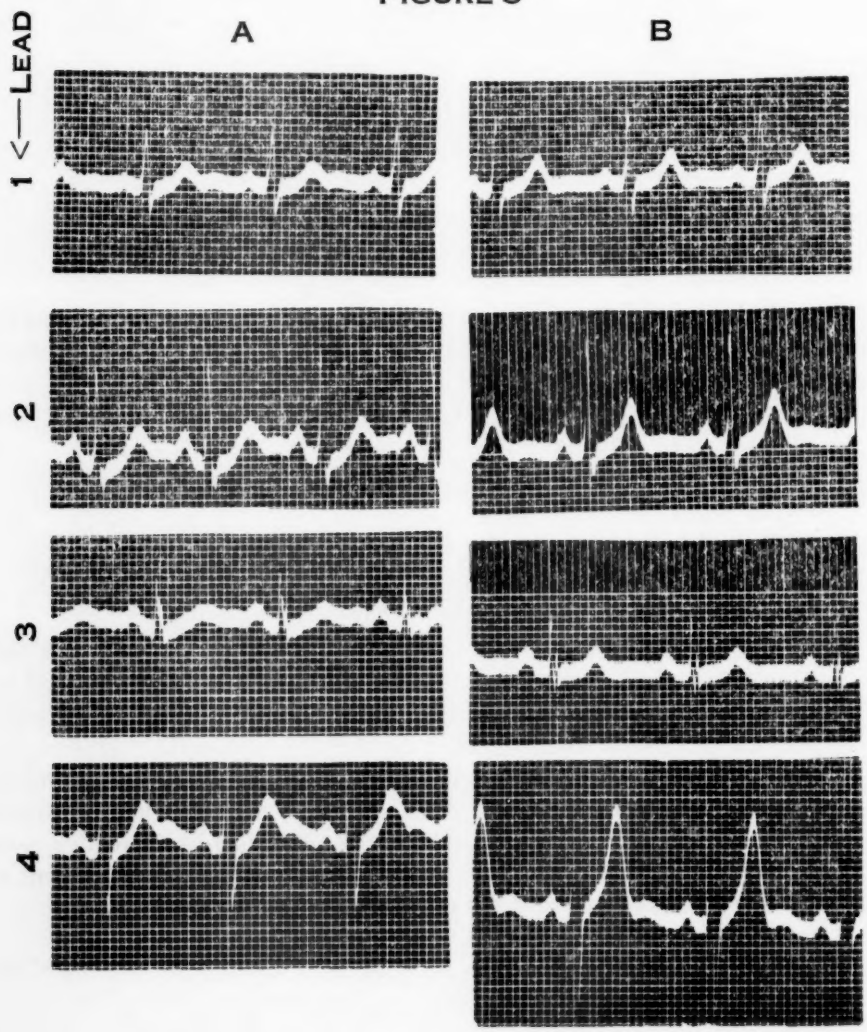
On February 20, gynecological examination disclosed the fact that this patient was pregnant. She was admitted to the University Hospital on the gynecological service, and the pregnancy was terminated. Her stage of pregnancy was estimated at approximately three months. During the period of hospitalization, no dyspnea, edema or tachycardia were noted. In August, 1940, approximately a year and a half after her last treatment with metrazol, she was examined again. She gave no history of dyspnoea or edema since her last interview. No edema or dyspnea was present at this time. The pulse rate was 140 during the examination.

Electrocardiogram, August 29, 1940. (Figure 2C.)
Rate: A-125 V-125
Rhythm: Regular

P. R.: 0.16
QRS: 0.08
Reading: Depressed $ST_{1\,2\,3}$; low T_1; deep S_3
Changes consistent with slight left ventricular strain

Case 3. R. E. R., white male, aged 19 years, admitted to the University Hospital on February 8, 1939, with typical symptoms of schizophrenia, catatonic type. Physical, neurologic and laboratory examinations revealed nothing significant.

FIGURE 3



Electrocardiogram, February 8, 1939 (prior to treatment.) (Fig. 3A.)
Rate: A-100 V-100
Rhythm: Regular
P. R.: 0.14
QRS: 0.08
Reading: U waves are presest. $ST_{1\,2\,3}$ are depressed
There is slight slurring of QRS waves. QRS_3 is notched
Impression:
Normal sinus mechanism
Slightly abnormal ST segment
Probably physiologic curve

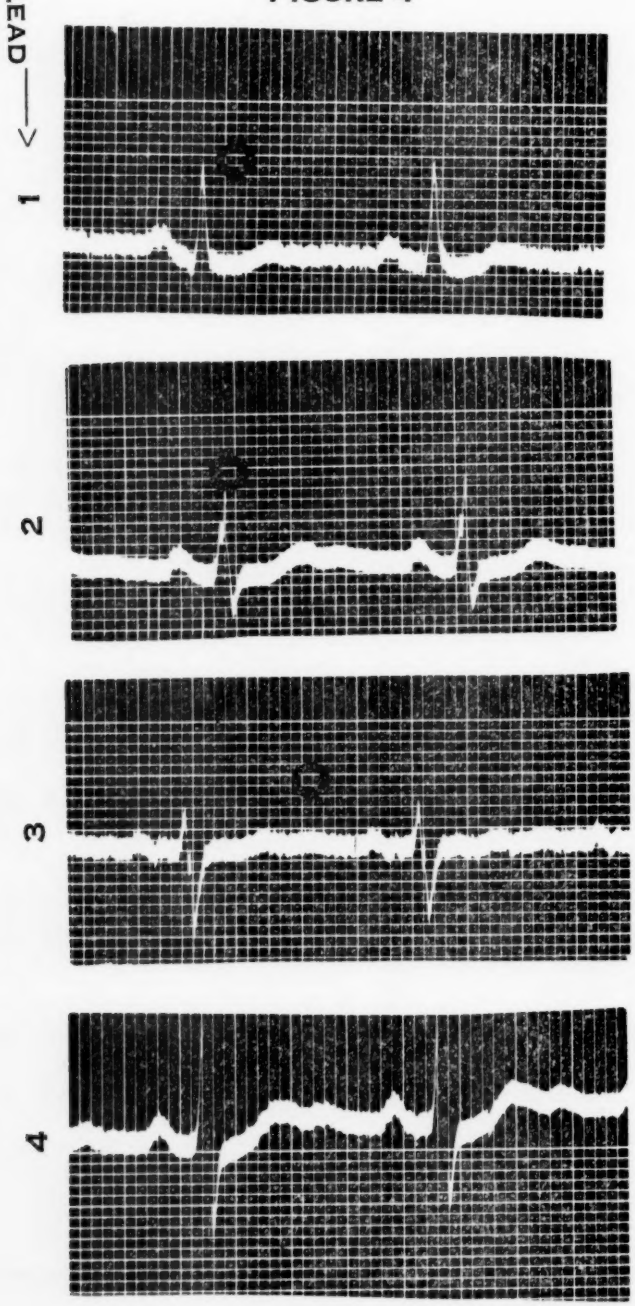
Metrazol therapy was begun on February 9, and continued through February 26, with 14 injections given and eight major seizures occurring, the dosage varying from 4.5 cc. (first treatment) to 7 cc. (last treatment). On several occasions, unusual reactions were noted after the seizures. Twice (February 20 and 23) respiration failed to occur spontaneously. Cyanosis became extreme. Artificial respiration and oxygen by nasal tube were administered while cyanosis increased for two minutes before breathing began. On both of these occasions the patient presented a very convincing appearance of a cadaver; and on the second, of a cadaver that had stood for some time. On February 26 (eighth convulsion), cyanosis was again extreme. Respiration was regained spontaneously, but during the first minute after the seizure the radial pulse rate fell to nine beats per minute. Although the rate increased, it remained slow (18-40) for several minutes and was, during this time, very feeble.

Electrocardiogram, February 26, 1939 (taken a few hours after the seizure. (Figure 3B.)
Rate: A-85 V-85
Rhythm: Regular
P. R.: 0.13
QRS: 0.06
Reading: The T waves in all leads are of greater voltage than in the previous electrocardiogram
Impression:
Normal sinus mechanism
Physiologic curve

The patient recovered from his psychosis and showed no evidence of cardiovascular damage.

Case 4. R. McC., white male, 57 years of age, admitted to the University Hospital, April 12, 1940, with an involution psychosis characterized by

FIGURE 4



paranoid delusions, agitation, despair, and ideas of guilt and unworthiness. Mild peripheral and retinal arteriosclerosis were noted. General physical, neurologic and laboratory examinations (including lumbar puncture) showed nothing significant.

Electrocardiogram, April 16, 1940. (Figure 4.)
Rate: A-70 V-70
Rhythm: Regular
P. R.: 0.16
QRS: 0.10
Reading: Depressed $ST_{1\,2\,3\,4}$; blunt T_4; deep $S_{2\,3}$; ventricular premature beat in L_3
Impression:
Normal sinus mechanism
Myocardial damage, moderate
Left axis deviation, slight
Ventricular premature beat in L_3

This patient was treated with insulin and metrazol on alternate days from April 18 to May 1. Five complete and typical convulsive reactions with metrazol were obtained, and full coma was reached twice. He was dismissed May 1, 1940, slightly improved in his personality status and without any sign or symptom of cardiovascular disorder. Approximately 10 hours after leaving the hospital, weakness and dyspnea suddenly became manifest. The patient found himself unable to stand. Cyanosis was observed. These signs and symptoms progressed until death occurred approximately an hour later.

An autopsy revealed the presence of massive pulmonary infarction and of a large antemortem thrombus in the right atrium of the heart. Apparently part of the thrombus which had formed in the heart became detached after the patient had left the hospital and had at this time produced sudden pulmonary infarction which was almost certainly the direct cause of death. Why the thrombus should have formed in the right atrium cannot be positively answered. Since auricular fibrillation has been observed to occur transiently after metrazol therapy[9, 11, 13] and insulin therapy[3] one might suspect that a temporary period of fibrillation occurred unobserved in this patient. If this did occur, it seems possible that the thrombus formation was related to the fibrillation. After normal rhythm has been restored it is not uncommon for such thrombi to become detached and cause fatal infarction. At autopsy, a moderate atherosclerosis of the coronary vessels with slight narrowing of the lumen was also noted.

Case 5. M. T., colored female, 25 years of age (approximate) admitted to the University Hospital, March 25, 1940, emotionally disturbed, hyper-

active and presenting typical features of schizophrenia. General physical, neurologic, laboratory and roentgenologic examinations revealed nothing significant except tachycardia which was interpreted as a product of an emotional disturbance.

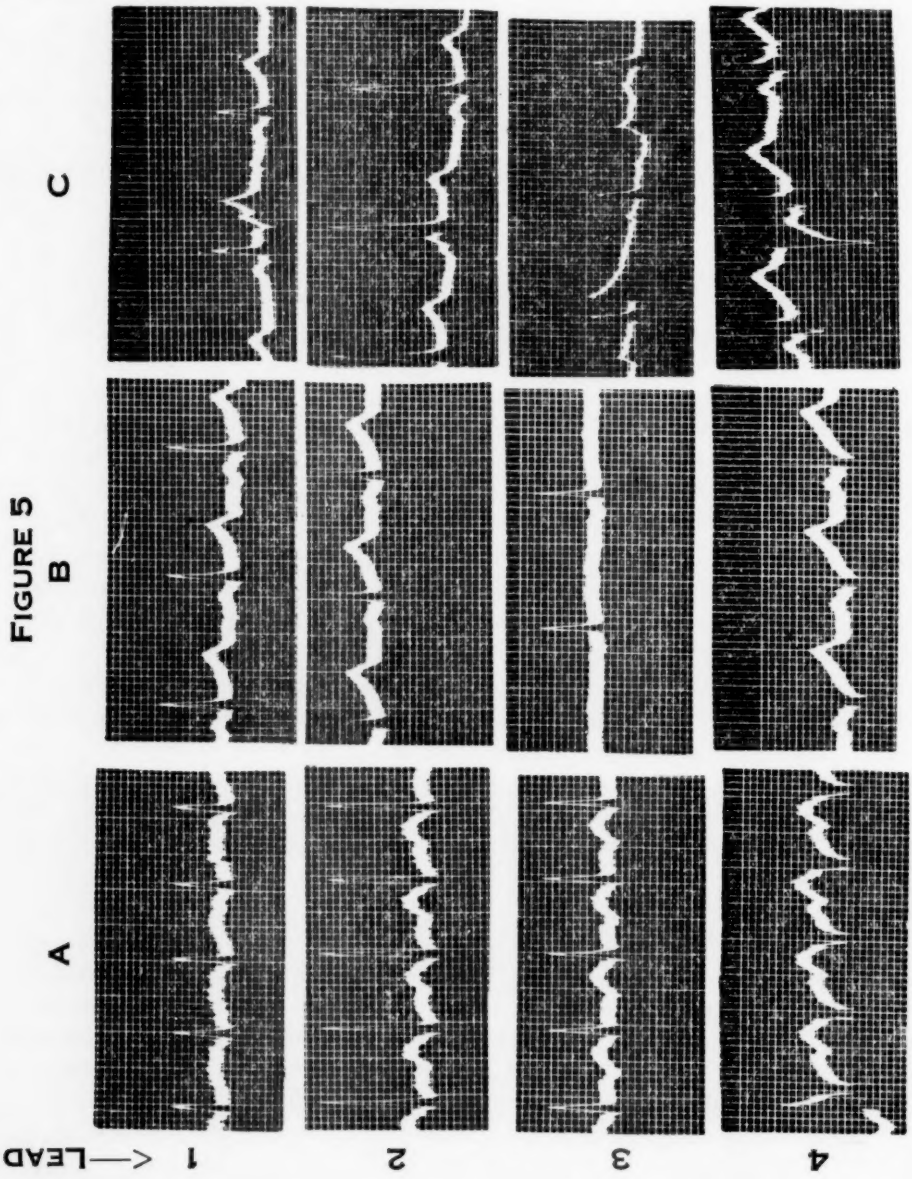


FIGURE 5

Electrocardiogram, March 25, 1940. (Figure 5A.)
Rate: A-145 V-145
Rhythm: Regular
P. R.: 0.18
QRS: 0.07
Reading: Low T_1; slight late inversion; T_2 and T_3; $Q_4 = -4$; T_4 diphasic, tending to late inversion; slight slurring of QRS waves
Impression:
S. A. Tachycardia
Myocardial damage, severe

Despite the electrocardiographic report, metrazol therapy was administered from March 25 to April 15, 1940, 11 grand mal seizures being obtained with 11 injections varying from 3.5 cc. to 5.4 cc.

Several hours after the third seizure a second electrocardiogram was made—on March 30, 1940.

Electrocardiogram, March 30, 1940. (Figure 5B.)
Rate: A-86 V-86
Rhythm: Regular
P. R.: 0.14
QRS: 0.06
Reading: Low T_3
Impression: Physiologic curve

A third electrocardiogram made on April 13, 1940, and a fourth made on May 19, 1940, (Figure 5C) showed no important changes from the second and were regarded as normal by the cardiologist. No clinical evidence of cardiovascular disorder has been noted.

Comment

The electrocardiographic changes and other cardiovascular reactions observed in these patients, though all somewhat unusual in our experience, showed little in common. Case 1, whose severe and persistent cyanosis implied serious cardiac embarrassment, was found five days after the last metrazol treatment to have developed electrocardiographic evidence of slight myocardial damage. His tracings improved, however, and though slight changes of questionable significance were noted 12 days after the last treatment, 10 months later no evidence of abnormality existed. The fact that this patient received insulin, though not in amounts sufficient to produce full coma, must not be forgotten.

Case 2, who showed somewhat more cyanosis and respiratory difficulty after seizures than the usual patient, was found to have a normal electrocardiogram after receiving 10 convulsive doses of metrazol. Five days after being dismissed from the hospital, however, she developed clinical signs and symptoms strongly suggestive of heart failure. At this time, the electrocardiogram showed changes probably dependent on the tachycardia but which the cardiologist believed might indicate cardiac pathology. The fact that this patient was in an early stage of pregnancy must not be overlooked. A year and a half later, tachycardia was still present, but during the intervening time there had been no dyspnea or edema and there were no other clinical signs of cardiovascular disorder. A final electrocardiogram a year and a half after her last metrazol treatment showed variations consistent with slight organic damage. The last electrocardiogram is not unlike those described by Rathnell[15] who, after a year, found changes in the S-T segment and the T wave.

Case 3 before treatment showed slight electrocardiographic variations from the normal but no change definitely indicative of cardiovascular disorder. Immediately after the convulsions, he developed alarming pulse changes and cyanosis. During the first minute after one of the convulsions a condition approaching cardiac standstill, as noted by Forschbach,[12] was observed. This patient, despite his disquieting reactions, showed an improvement in the electrocardiogram after treatment.

Case 4, who received insulin in sufficient dosage to produce full coma, as well as metrazol, died from massive pulmonary embolism. There is little doubt that the embolus came from an earlier thrombus in the right atrium of the heart. Evidence of moderate myocardial damage and other changes were noted in the electrocardiogram made prior to the treatment. The assumption that a period of temporary fibrillation occurred unobserved, either during insulin treatment or after metrazol, offers the most likely explanation of the thrombus in the heart, and therefore, indirectly, of this patient's death.

Case 5, prior to treatment, showed electrocardiographic evidence of severe myocardial damage. During treatment with metrazol the electrocardiogram became normal and has remained so in subse-

quent readings, the last of which was made six weeks afterward. No clinical evidence of cardiovascular disorder or damage was noted during or after the treatment. The observations in this case as well as in Case 3 might be regarded as confirmatory of the opinion expressed by Messinger and Moros[17] who concluded that the effects of metrazol therapy tended to improve heart action.

Summary and Conclusions

1. In a survey of the available literature instances of temporary arrhythmias and other electrocardiographic changes are frequently recorded. Conclusive evidence is not found of permanent myocardial damage or other serious change in patients with normal cardiovascular systems at the beginning of treatment. Evidence of improvement is offered by some observers.[17]

2. In the five cases discussed here, alarming clinical signs immediately after the convulsions were noted in some. Definite electrocardiographic evidence of myocardial damage, noted in one case five days after the last seizure, had disappeared entirely 10 months later. One case, in which clinical signs of moderate cardiac decompensation developed several days after the termination of treatment, now, after 18 months, still shows tachycardia and electrocardiographic changes consistent with slight myocardial damage, but has no clinical signs of decompensation. In one case, the patient died within 24 hours after leaving the hospital from pulmonary embolism which may have been related to auricular fibrillation produced by metrazol or insulin. Two cases showed definite electrocardiographic evidence of improvement. In one of these, the apparent improvement was striking.

3. After seizures induced by metrazol, cardiovascular reactions may occur which suggest dangerous effects. The desirability of terminating treatment under such circumstances should be considered. As judged by electrocardiograms a few cases appear to show improvement, which, in rare instances, may be extremely impressive. Whether the favorable electrocardiographic changes represent real improvement in the heart or merely a lack of finality in current methods of interpretation, is a question not to be answered here.

4. It would appear that extreme or persistent cyanosis and dyspnea may indicate effects on the heart capable of producing significant pathological changes. It has been our experience that atropine administered prior to the metrazol therapy tends to prevent the accumulation of excessive secretions in the air passages and to diminish dyspnea. In our cases cyanosis has also been less frequently persistent or marked when atropine was used. The administration of oxygen by an intranasal catheter, which is put in place during the tonic phase of the fit, has also appeared to reduce these complications. In occasional instances, such as the reaction described in Case 1, the writers believe that these precautionary measures may have played an important part in preventing a fatal outcome. Therefore, their use routinely in metrazol therapy is advised.

5. These observations can scarcely be held to prove that either immediately fatal cardiac disturbances or permanent myocardial damage is produced in patients free from cardiovascular pathology prior to treatment. The possibility of both of these effects is, however, suggested. The writers' experience indicates that if such effects occur they must be rare and should certainly not discredit the use of metrazol in serious personality disorders. In other instances, it would appear that metrazol therapy may cause the disappearance of electrocardiographic findings suggesting serious cardiac disease.

University of Georgia
School of Medicine
Augusta, Ga.

REFERENCES

1. Friedman, E.: The irritative treatment of schizophrenia. Am. J. Psychiat., 94:355-372.
2. Hadorn, W.: Insulin and the circulation. Am. J. Psychiat. (supp.), 94:89-96.
3. Bond, E. D., Hughes, J., and Flaherty, J. A.: Results and observations on the insulin shock treatment of schizophrenia. Am. J. Psychiat., 96:317-326.
4. Reese, H. H., Vander Veer, A. H., and Wedge, A. H.: The effect of induced metrazol convulsions on schizophrenic patients. J. Nerv. and Ment. Dis., 87:570-583.
5. von Meduna, L.: Die Konvulsionstherapie der Schizophrenie. Halle, Carl Marhold, 1937.
6. Hayman, Max, and Brody, Morris W.: Metrazol therapy in schizophrenia—Report of a fatal case with autopsy. J. A. M. A., 112:310, January 28, 1939.
7. Geraudel, Emile: Electrocardiographic studies of cardiazol (metrazol) treatment of schizophrenia. Arch. des. Maladies du Coeur, 47:311, 1938.

8. In discussion of: Kagan, J., and Fingert, H.: Presentation of metrazol treated cases. J. Nerv. and Ment. Dis., 88-69, July, 1938.

9. Dick, A., and McAdam, W.: Cardiac complications in cardiazol (metrazol) treatment: Observations in four cases. J. Ment. Sci., 34:999, November, 1938.

10. Pellens, M.: Electrocardiographic studies of patients receiving convulsant doses of metrazol in the treatment of schizophrenia. PSYCHIAT. QUART., 12:722, October, 1938.

11. McAdam, W.: Some cardiac accidents associated with cardiazol (metrazol) convulsion therapy. Glasgow Med. J., 12:221, November, 1938. (Abst. in J. A. M. A., 112:89, January 7, 1939.)

12. Forschbach, Gerhard: Electrocardiographic studies during the course of metrazol treatment of schizophrenia. Ztschr. f. d. ges. Neurol. u. Psychiat., 164:722, February, 1939.

13. Strecker, E. A., Alpers, B. J., Flaherty, J. A., and Hughes, J.: Experimental and clinical study of effects of metrazol convulsions. Arch. Neurol. and Psychiat., 41:996, May, 1939.

14. Gross, M., and Gross-May, G.: Accidents in course of metrazol therapy of schizophrenic patients. J. Belge d. Neur. et d. Psych., 39:336, May, 1939.

15. Rathnell, Thomas K.: Electrocardiographic studies following use of metrazol. Proceedings of the meeting of the Philadelphia Psychiatric Society, March 10, 1939. Arch. Neurol. and Psychiat., 42:778, October, 1939.

16. Orenstein, Lee L.: Physiologic observations following induced convulsions. N. Y. S. J. Med., 39:1921, October 15, 1939.

17. Messinger, E., and Moros, N.: Cardiovascular effects of large doses of metrazol as employed in the treatment of schizophrenia. Annals Int. Med., 13:1184, January, 1940.

18. Levine, Harold, Piltz, George F., and Reznikoff, Leon: Electrocardiographic studies in metrazol shock therapy. Report of electrocardiographic findings obtained in the treatment of 50 schizophrenic patients with convulsive shock induced by metrazol. Am. J. Med. Sci., 199:201, February, 1940.

19. Bennett, A. E.: Metrazol convulsive shock therapy in affective psychoses. Am. J. Med. Sci., 198:695, November, 1939.

THE ELECTROCARDIOGRAM IN METRAZOL THERAPY*

BY LOUIS WENDER, M. D., AND ABRAHAM JEZER, M. D.

This preliminary report deals with a study made at Hastings Hillside Hospital of 75 injection treatments in patients suffering from affective disorders who received metrazol in convulsive doses. Metrazol in small doses has been recommended for use as a circulatory and as a respiratory stimulant. Meduna[1] introduced it in large doses as a convulsive agent in the treatment of the psychoses. This report deals with the effects of metrazol upon the circulatory system during metrazol shock therapy, as recorded electrocardiographically.

Metrazol was administered intravenously in increasing doses, usually with an initial dose of 3.5 cc. Following the injection an increased respiration with an increased pulse rate is observed. This is followed in close succession by a period of apnea, a tonic spasm and then a clonic seizure which lasts for a period of 15 to 40 seconds. This is followed by a period of apnea which lasts from a few seconds to a minute. During this apneic period, cyanosis and marked vasomotor disturbance are observed. The pulse at times becomes hardly perceptible and on many occasions disappears for a half-minute to a minute. Shortly thereafter, respiration becomes normal, the pulse becomes bounding.

Because of these clinical observations, it was considered advisable that an electrocardiogram be made for each patient, so that the effects of this drug on the heart rate, rhythm and conduction mechanism might be more fully determined. Records were obtained before, during and after the convulsion.

The amount given varied from 3½ cc. to 13 cc. On a few occasions, a second dose was administered two to three minutes after the first injection, when no convulsion followed the first dose. On one occasion a total dosage of 27 cc. was given to one patient in

*Read before the New York Neurological Society, December 3, 1940.

three doses in a period of five minutes. Treatments were given three times each week.

Electrocardiograms were taken and repeated observations were made on the same patients during a number of treatments. In several cases, intervals elapsed between the tests reported.

The age variation of the group ranged from 15 to 48 years. With the exception of two patients, who were 15 and 17, they were equally divided among the third, fourth and fifth decades. The sexes were also about equally divided.

The electrocardiograms obtained in these observations were analyzed from three points of view. These are: 1. Influence on the ventricular complexes. 2. Influence on the rhythm. 3. Influence on the rate of the heart.

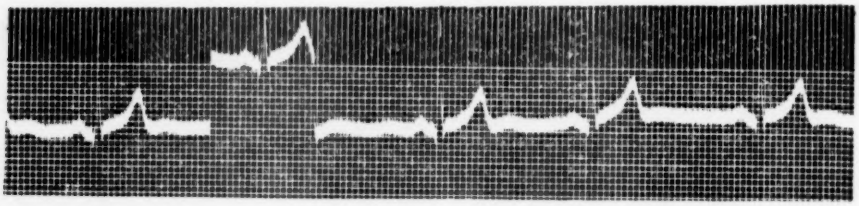
1. *Influence of the Drug on the Ventricular Musculature.* These observations were obtained in the preliminary injections when no convulsions followed. Particular attention was paid to the ventricular complexes and to the changes in the R-T and S-T segments, because important changes in the ventricular musculature would be translated in the electrocardiogram in changes in the ventricular complexes and in the R-T segment.

The changes in the ventricular complexes which followed the convulsion may be the result, either of the action of the drug, or of the effect of the convulsion, or of the effects of both factors.

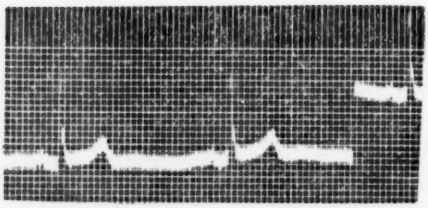
2. *Changes in Rhythm.* The changes of the rhythmicity of the heart may play an important rôle in deciding on the advisability of administering this drug to cardiacs. Changes in rhythm were observed, and their courses were followed from the time of injection of this drug until the return of the basic heart rhythm.

3. *Influence on the rate of the heart beat.* The increase of the heart rate beyond normal limits may not be important if the increase persists for only a short period; its persistence for longer periods may militate against the use of metrazol in cardiacs.

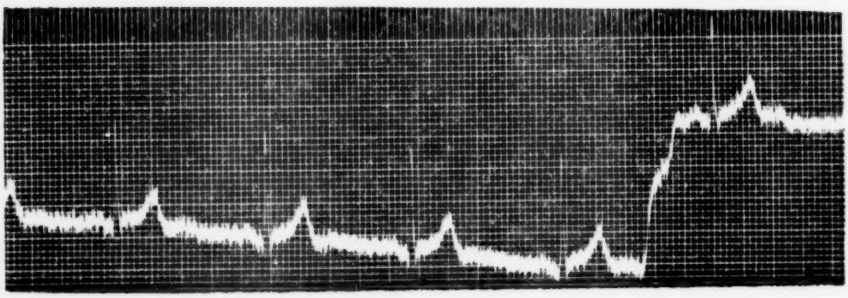
Figure 1. The Effects of Metrazol on the Form of the Electrocardiogram.
Figure 1-A.



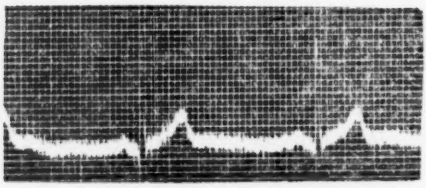
Control



One minute after metrazol



Two minutes after metrazol



Second injection of metrazol

Fig. 1-A. There is a very slight and transient R-T elevation in the record obtained one minute after the injection of metrazol. In the electrocardiogram of the second minute after the injection, the ventricular complexes have returned to their basic form.

A second injection of 3.5 cc. of metrazol was then given and again no change in the form of the ventricular complexes was noted before the convulsion set in.

Figure 1-B.

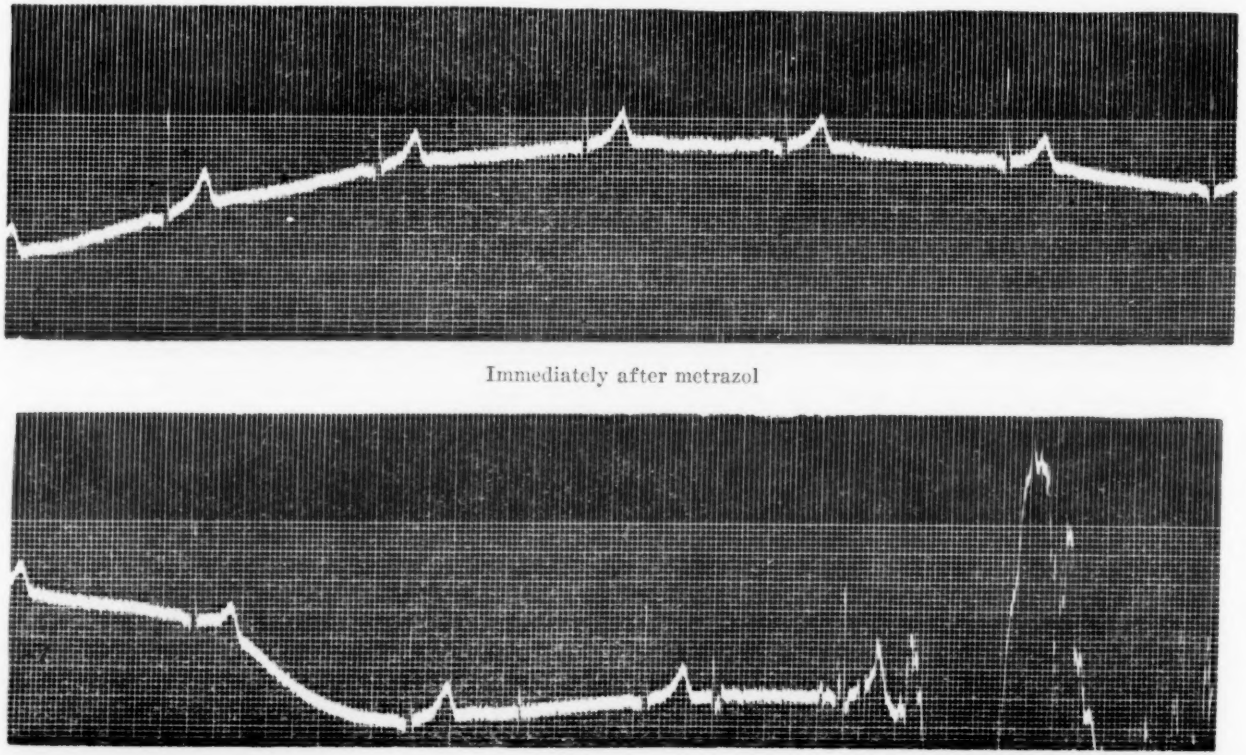
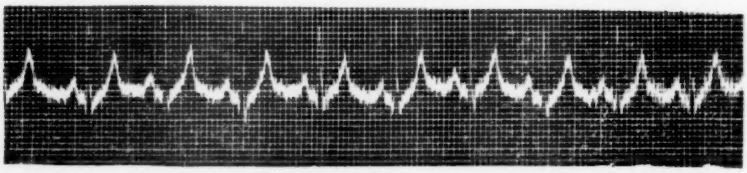
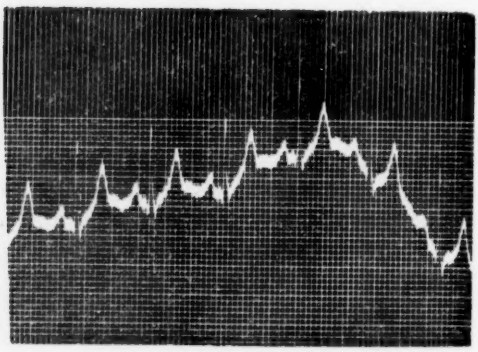


Fig. 1-B. A slowing of the heart from a rate of 50 to a rate of 38 beats per minute is shown for about 20 seconds just preceding the onset of the convulsion. Subsequently, another injection was given to the same patient. A control was taken before the injection of 5 cc. of metrazol. The rate is 70 beats per minute.

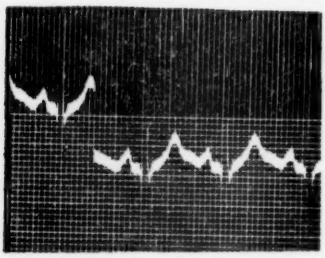
Figure 1-C.



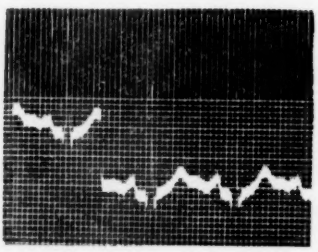
One-half minute after convulsion



Two minutes after convulsion



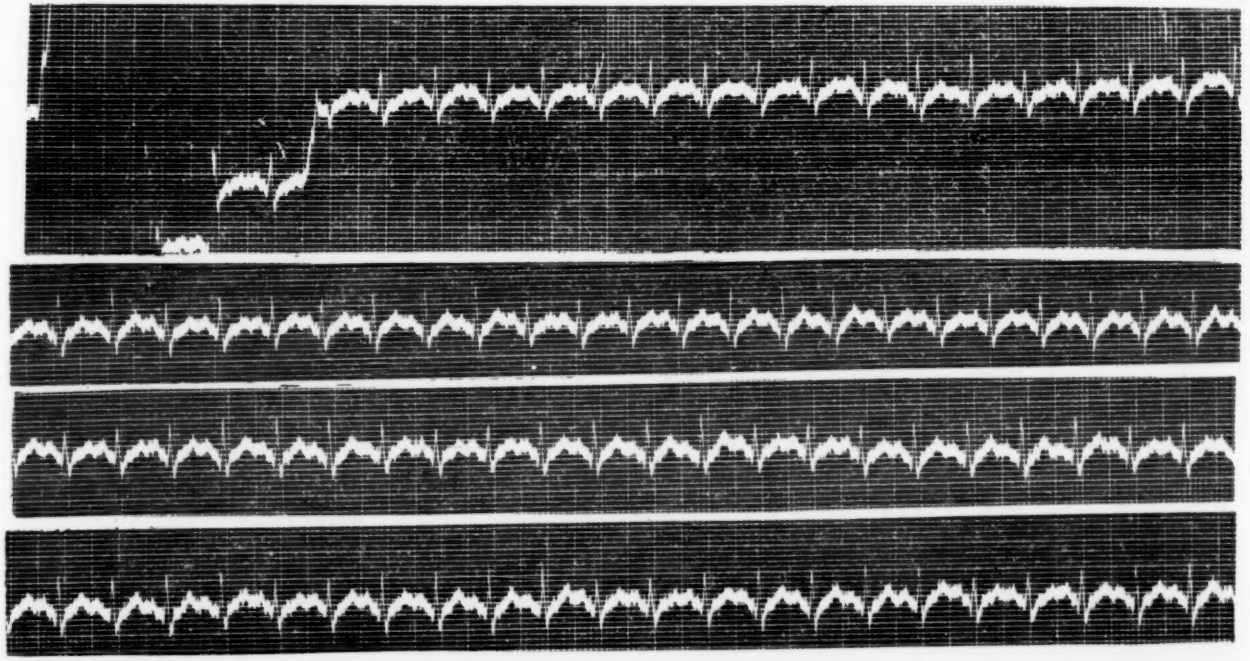
Seven minutes after convulsion



Ten minutes after convulsion

Fig. 1-C. Immediately after the injection the form and shape of the ventricular complexes are unchanged, but the rate is increased to 115 beats per minute. About 15 seconds after the injection, the S-T segment is seen to be very slightly depressed.

Figure 2. The Electrocardiogram of the Convulsion

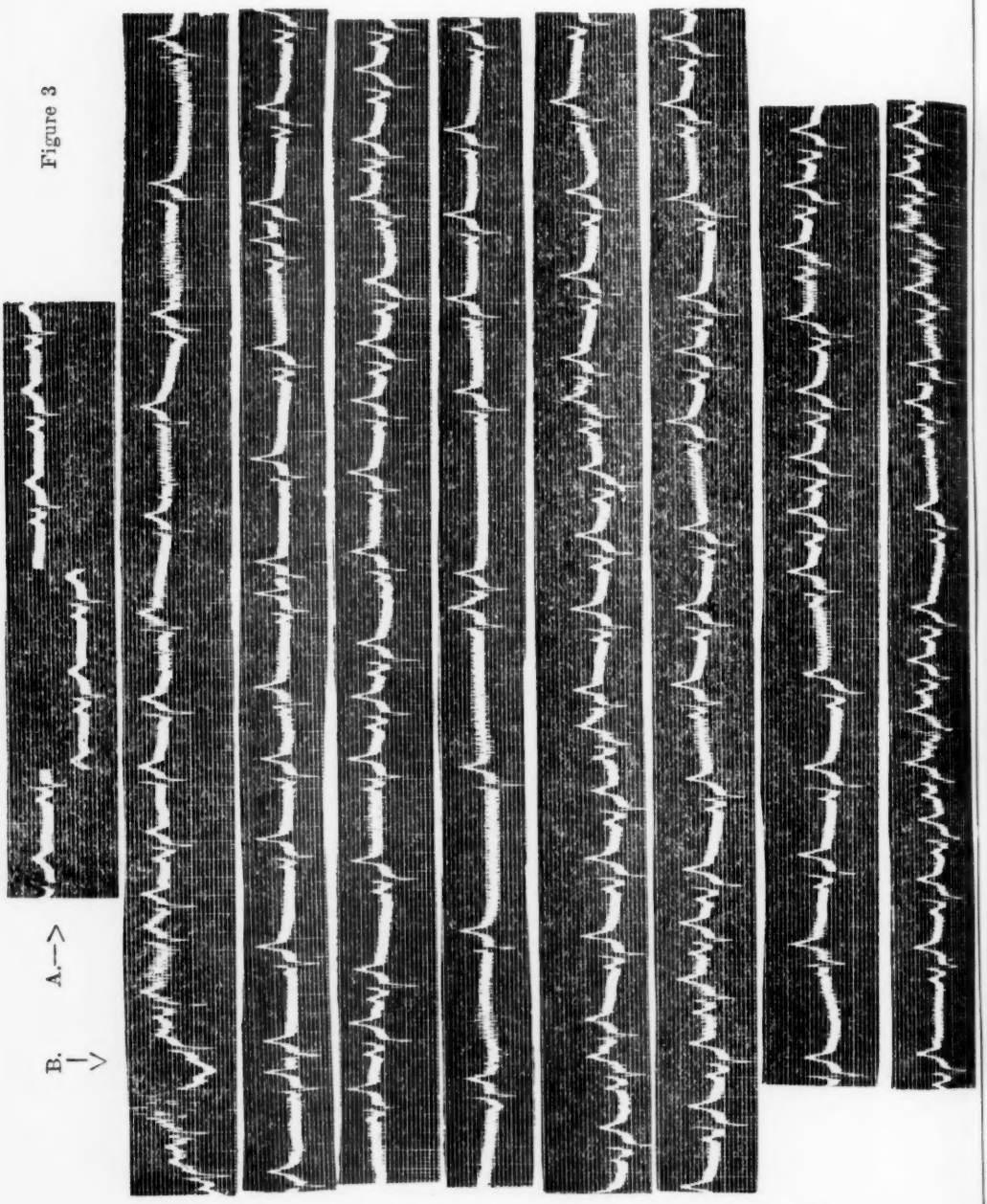


Immediately after convulsion

The heart rate in this instance increased from a control rate of 100 beats per minute to a rate of 150 beats per minute, immediately after the convulsion stopped. This rate gradually slowed, so that after seven or eight minutes the rate approached the normal. This is the usual method by which the heart returns to its basic mechanism. The ventricular complexes are unaltered except for a slight depression of the RS-T segment.

OCT.—1941—E

Figure 3



A. Control. B. Immediately after convulsion. (Continuous strip.)

Discussion

The writers have demonstrated that the effect of metrazol on the ventricular complexes is transient and is of no clinical importance. No additional changes were noted in patients who were observed during the first treatment or during subsequent treatments. Repeated injections, in one instance three successive doses, failed to produce any changes worthy of note. Similar observations were reported by Bennett,[2] McAdam,[3] Forschbach,[4] and Orenstein.[5].

The writers have also demonstrated many changes in rhythm. These changes in rhythm are usually sinus arrhythmias with varying degrees of sinus node depression. These findings were observed in more than 75 per cent of the cases.

Changes following the marked depression of the sinus pacemaker and the release of the auriculoventricular nodal pacemaker were present as fleeting findings in about half of the group. A small number of these showed a persistence of this arrhythmia for periods up to two to three minutes. Usually, the nodal rhythm alternated with the sinus arrhythmia, resulting in a gross irregularity of the heart rhythm.

In two observations the arrhythmia was the result of partial heart block (A-V block). In one instance complete ventricular standstill for 20 seconds followed soon after the convulsion. A similar observation was reported by Forschbach.[4]

These arrhythmias are the so-called "vagal arrhythmias" and, in other instances, have been observed in pneumoencephalography and following subarachnoid hemorrhage and cerebral accidents, where the heart has been normal.

Figure 3. Changes in Rhythm Following the Convulsion.

Immediately following the convulsion, the heart rate has slowed; the P waves disappear from time to time, and at other times are superimposed on the ventricular complexes. This rhythm is due to a depression of the sinus pacemaker and the assumption of the heart rhythm by the auriculoventricular node. After eight seconds, the sinus pacemaker again controls the heart rhythm. This rhythm is interrupted on occasion by a disruption of the normal sequence of conduction from the auricles to the ventricles (end of strip 2 and early strip 3). A marked phasic arrhythmia then follows and continues as the basic rhythm of the heart for the next eight to 10 minutes.

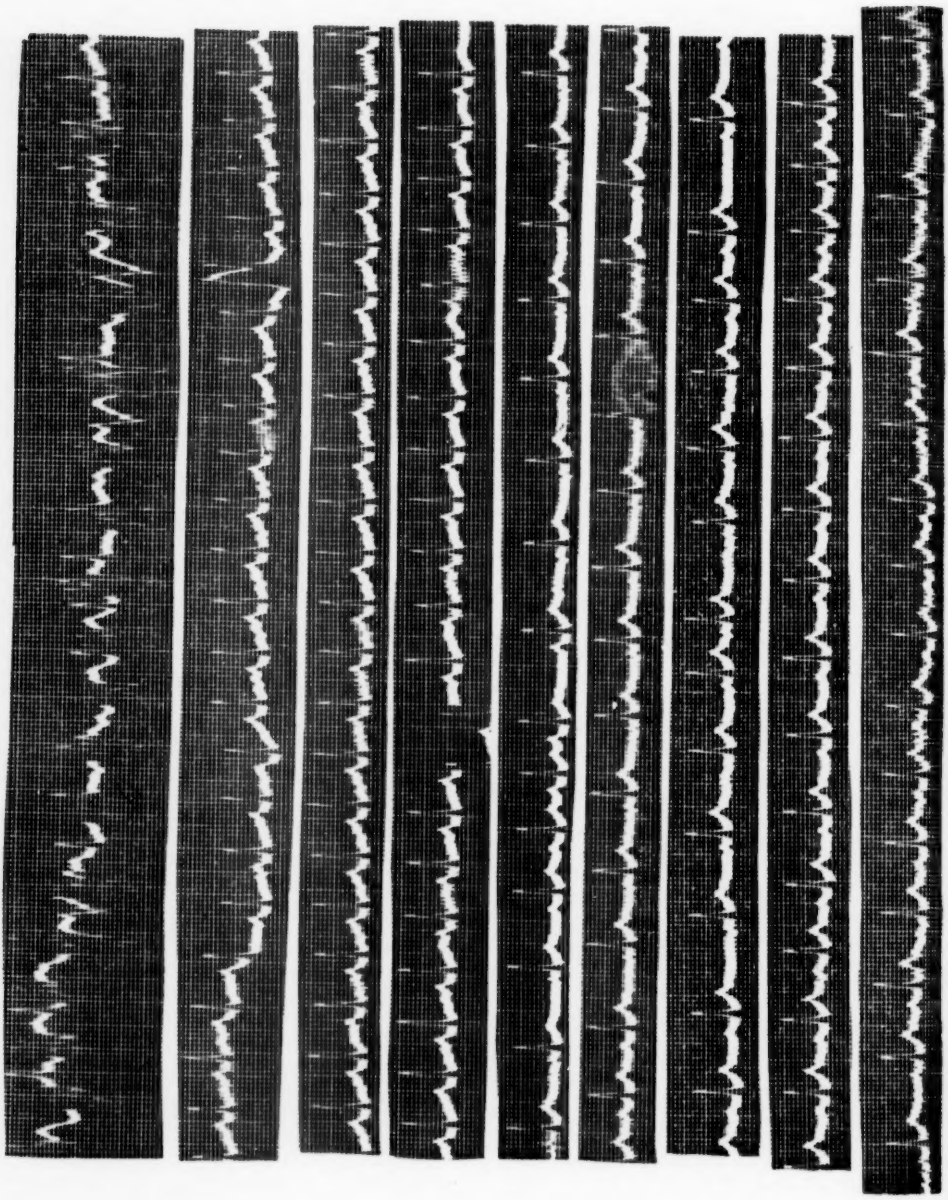


Figure 4.

It is also important to observe that the same patient reacts on one occasion by the production of a sinus block with marked sinus arrhythmia and nodal beats, and on another occasion by simple increase in the heart rate.

Since there are no changes in the ventricular complexes following the administration of this drug, and since the arrhythmias only follow the convulsion, it is the writers' opinion that the arrhythmias are not cardiac in origin but are the result of the effects of the extrinsic nervous mechanism on the pacemakers of the heart. These effects are probably the result of autonomic nervous system imbalance, with the parasympathetic influence predominating for several minutes after the convulsion.

The rate of the heart usually increases to a level of 135 to 180 beats per minute for one-half to one minute following the convulsion and then gradually falls to a more basic level after five to 10 minutes. The amount of the drug given could not be correlated by the writers with the increase in the rate of the heart or with changes in the rhythm.

Conclusions

1. The writers have demonstrated that the repeated administration of metrazol does not produce any important change in the ventricular complex. There is a transient change in the S-T segment which is the result of the increase in the heart rate and which usually disappears when the rate falls to a more basic level.

2. There are no changes of any clinical importance which follow the injection of metrazol where no convulsion occurs.

3. In patients in whom metrazol has produced a convulsion there are three distinct types of arrhythmias which may be pro-

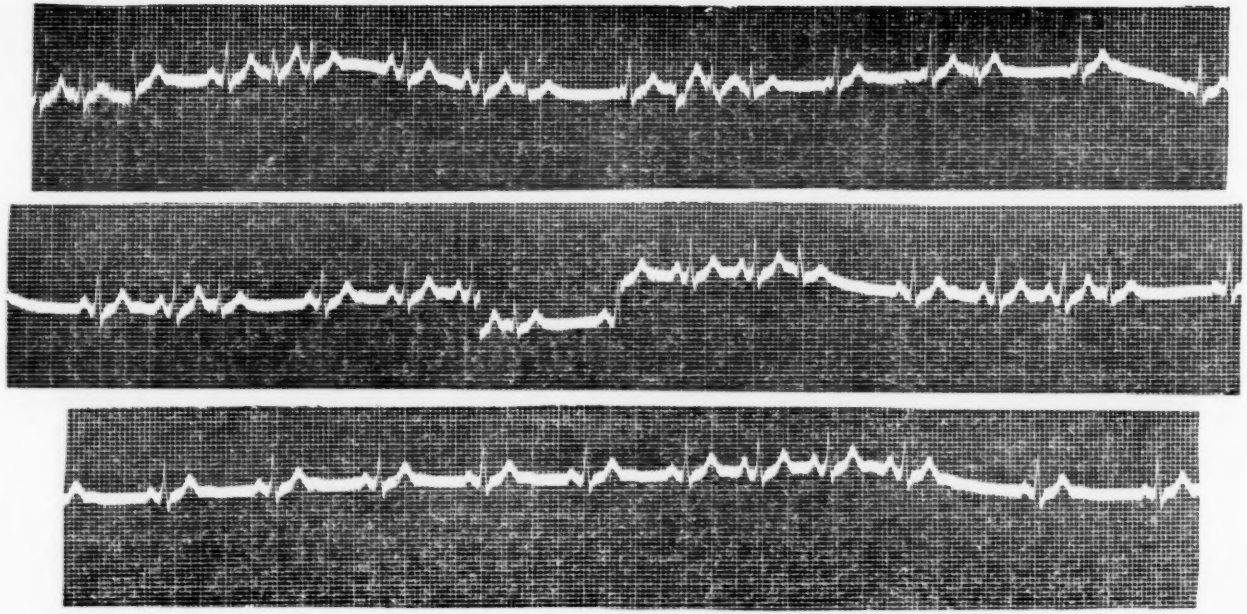
---

Figure 4. Changes in Rhythm Following the Convulsion.

This record shows a similar depression of the sinus pacemaker and the assumption of the control of the heart rhythm by the auriculoventricular pacemaker for a period of about 10 seconds, half a minute after the convulsion has ended. Then there follows an alternation of the control of the heart by the sinu-auricular pacemaker and by the auriculoventricular pacemaker. When the sinus pacemaker initiates impulses at a rate which is more than 60 beats per minute, its influence predominates. When this normal pacemaker is depressed so that its rate of impulse discharge falls below 60, then the auriculoventricular pacemaker takes control. This results in a marked and gross irregularity of the heart which cannot be differentiated clinically from auricular fibrillation.

Figure 5.

One minute after convulsion



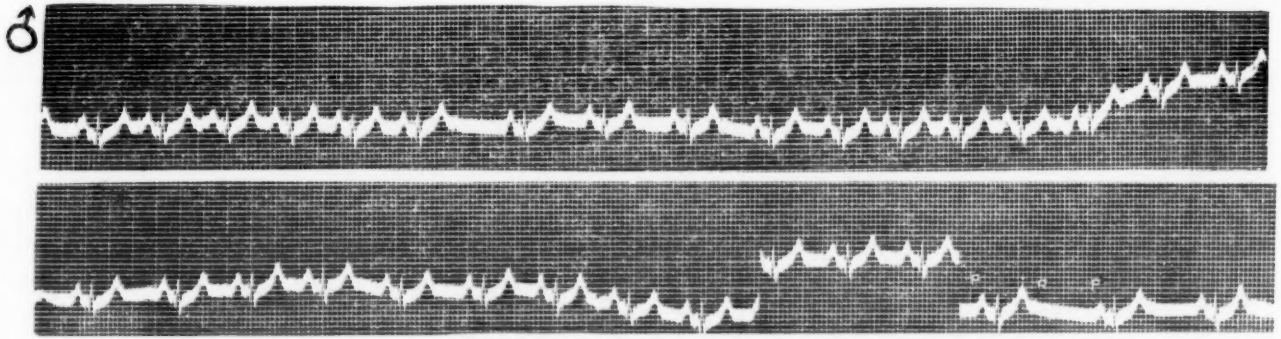
Lead 2—Continuous strip

Figure 5. Changes in Rhythm Following the Convulsion.

This record shows the presence of multiple premature auricular beats one minute after the convulsion. The premature beats arise individually, in pairs, or in groups of three. Immediately following such a group of premature beats, the sinus pacemaker is again depressed, and auriculoventricular nodal rhythm follows.

Figure 6.

Two minutes after convulsion



Five minutes after convulsion

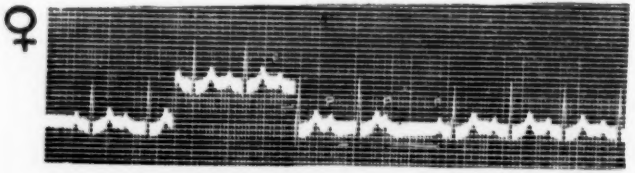


Figure 6. Changes in Rhythm Following the Convulsion.

Two observations in two individual patients, both showing depression of conduction in the auriculoventricular node and resulting in partial heart block.

duced. (a.) The most usual type is the phasic sinus arrhythmia. This may or may not be accompanied by periods of sinus block. (b.) A gross irregularity in rhythm produced by the alternation of the control of the rhythm of the heart by the normal sinus pacemaker and the auriculoventricular nodal pacemaker. This A-V nodal rhythm follows sinus arrest. (c.) Rarely, heart block follows the convulsion induced by this drug.

4. These arrhythmias are all manifestations of increased vagal influence and can be reproduced in susceptible humans by the application of carotid sinus pressure, by the administration of digitalis, or by inducing changes in intracranial pressure.

5. These changes are transient, when produced by the convulsion following the administration of metrazol and have proved to be of no clinical importance in the writers' series.

6. There is no contraindication to the use of metrazol from the cardiovascular aspect in a patient with a normal heart or in a patient whose heart is slightly damaged.

7. These observations definitely show that sinu-auricular block or sinus arrest or sinus arrhythmia or auriculoventricular A-V nodal rhythm and, occasionally, heart block may be the results of changes in the central nervous system rather than changes in the heart muscle.

Hastings Hillside Hospital
Hastings-on-Hudson, N. Y.

REFERENCES

1. von Meduna, L.: Convulsive therapy of schizophrenia. Psych.-Neurol., Wchnschr., 37:317, July, 1935.
2. Bennett, A. E.: Metrazol convulsive shock therapy in affective psychoses. Am. J. Med. Sci., 198:695, November, 1939.
3. McAdam, W.: Some cardiac accidents associated with cardiazol convulsive therapy. Glasgow Med. J., 12:221, November, 1938.
4. Forschbach, Gerhard: Electrocardiographic studies during the course of metrazol treatment of schizophrenia. Ztschr. f. d. ges. Neurol. u. Psychiat., 164:722, February, 1939.
5. Orenstein, Leo L.: Physiologic observations following induced convulsions. N. Y. State J. Med., 39:1921, October 15, 1939.
6. Dick, A. and McAdam, W.: Cardiac complications in cardiazol treatment: Observations in four cases. J. Ment. Sci., 84:999, November, 1938.
7. Geraudel, Emile: Electrocardiographic studies of cardiazol treatment of schizophrenia. Arch. des Maladies du Coeur, 47:811, 1938.
8. Kagan, J. and Fingert, H.: Presentation of metrazol treated cases. J. Nerv. and Ment. Dis., 88:69, July, 1938.

SOME NUTRITIONAL ASPECTS OF BRAIN METABOLISM*

BY HERMAN WORTIS, M. D.

The brain is dependent for its normal functioning on a carbohydrate substrate,[1] an adequate supply of oxygen,[2] and various enzyme and coenzyme systems.[3] A disturbance in any of these constituents interferes with brain metabolism and therefore with brain function.

In addition, the nervous system is unique in containing a larger amount of lipids than any other tissue.[4] As a matter of fact, fatty substances comprise over one-half of the non-aqueous constituents of the brain, with the largest concentration naturally being present in the myelinated structures. Yet little is known concerning the metabolism and function of these lipid materials. It would seem likely, however, that a substance which comprises over one-half the solid material of the brain must have a very important function in that organ. We know, for example, that the failure of the body to oxidize properly the diaminophosphatide sphingomyelin is associated with the clinical syndrome of amaurotic familial idiocy. Obviously, many links in the chain remain to be forged, but even this simple fact brings something resembling therapeutic optimism to bear on a so-called "degenerative" condition leading to idiocy and early death.

Similarly, our knowledge of brain proteins is practically nil. Yet, many proteins have been isolated from brain tissue proper; and, according to Rose's classification,[5] the brain contains all the amino acids belonging to both the dispensable and indispensable groups, except cystine. We also know, for example, that a failure of the body properly to oxidize phenylpyruvic acid (a derivative of phenylalanine) is associated with a specific type of mental deficiency—phenlypyruvic oligophrenia.[6]

It would seem more profitable at present to evaluate those nutritional factors in which clinical medicine and psychiatry have kept pace with biochemistry, and, indeed, in several instances have shown the way.

*Presented at the New York Academy of Medicine, Society for Psychotherapy and Psychopathology, March 27, 1941.

Symptoms Due to Carbohydrate Lack

The brain, unlike other organs, oxidizes, for all practical purposes, only one foodstuff, dextrose.[1] Hence, as the available carbohydrate diminishes, the metabolism of the brain must necessarily diminish. This is clearly reflected in the diminished oxygen uptake by brain tissue during hypoglycemia.[7, 8] Himwich and his coworkers have, however, pointed out that this diminished metabolism does not indicate a lack of oxygen, but merely a diminished utilization of oxygen. As a matter of fact, the oxygen content of the brain may rise, owing to the diminished utilization of that gas.

Even in complete diabetes, when the rest of the body obtains most of its energy from the utilization of fat, the brain continues to burn carbohydrate.[9] This is particularly important, since the cerebral store of glycogen is a very meager one, and the brain is therefore dependent on circulating glucose to satisfy its energy demands.

From the work of Himwich and his coworkers,[10] it may further be seen that the effects of insulin hypoglycemia descend through the brain in a manner which may be determined phylogenetically. First, the activity of the cortex becomes suppressed, releasing the basal ganglia and the hypothalamus. The early symptoms disappear, in turn, and give rise to symptoms which may be associated with the midbrain; and, finally, the medullary centers are involved and death may result. Thus, the progress of the symptoms during hypoglycemia may be ascribed to a greater sensitivity of the newer portions of the brain to metabolic lack. As will be noted later, a similar progression of events may result when the brain is subjected to progressive anoxia.

As a matter of fact, even when the amount of glucose injected is adequate to supply the energy requirements of all parts of the brain, experiments on hypoglycemic animals reveal that the electrical activity of the hypothalamus is restored before that of the cortex.[11] Finally, it is obvious from both experimental and clinical data that if the hypoglycemia is continued too long, irreversible changes may set in, with permanent damage to brain functioning.

The clinical picture during hypoglycemia is exceedingly varied.[12] The prodromal symptoms usually consist of nervousness, perspira-

tion, pallor, tremor, sleepiness and salivation. These may clear up or progress in severity. The neurological manifestations begin with restlessness, diplopia and muscular twitching of the face, and then pass on through various stages of motor involvement, with the appearance of hemiplegias, Jacksonian attacks and tonic and clonic convulsions. Aphasia, hemianopia, disorders in sensation and coordination are common. The reflexes vary considerably, and there may be a Babinski toe sign. If the hypoglycemia is severe enough, the patient passes into stupor or coma.

Psychic disturbances vary from case to case, but form an integral part of the hypoglycemic picture. The early signs are anxieties, mild depressions and difficulties in concentration. Later the speech becomes thick, and there follow peculiar grimacing, unmotivated laughing and crying, hebetude, excitement, compulsions, fugues and hallucinatory states. Eventually, after varying degrees of confusion and clouding of the sensorium, consciousness is lost and stupor may result. Two brief case reports may best exemplify these changes.

Case 1. J. S., a 27-year-old male, was admitted to Bellevue Hospital because he had suddenly run beserk at a dinner party, had started to beat his wife and to call her a prostitute. He became uncontrollable, and two policemen were needed to subdue him sufficiently to get him into an ambulance. At the hospital, he was disoriented and confused; and he showed gross gaps in memory. He kept repeating, "That s. o. b., I'll get her yet." A history of diabetes was obtained from the wife. She said her husband had taken insulin before dinner, had had several drinks, and had complained of feeling very hungry. The dinner had been delayed, and she had noticed that he was becoming increasingly irritable and incoherent. This behavior continued, until the attack occurred which necessitated his hospitalization. His blood sugar was 30 mgms. per cent, and an injection of 50 cc. of 50 per cent glucose caused him to quiet down immediately, with a complete return to normal intellectual functioning. Both the patient and his wife insisted that their relationship was a normal one, although she did admit that he had accused her of infidelity three years ago, when she had embraced a friend of his at a party. When he was questioned, he denied that he could have had any possible motive for striking his

wife; and he never mentioned her name without assuring the physician of what a wonderful woman she was.

We may draw our own conclusions as to the true relations that existed between man and wife, but the fact remains that they were able to retain a fairly normal relationship until his cortex was deprived of its essential foodstuff.

Case 2. C. S., a 32-year-old white female, was admitted to the medical service of the Bellevue Psychiatric Hospital after having been in coma for 36 hours. The history obtained from her lover was admittedly incomplete, but the essential facts follow. The patient had been a chronic alcoholic for at least six years, and for several weeks prior to admission had been drinking heavily and eating very little food. Six months before her admission, a physician who examined her during an alcoholic debauch discovered sugar in her urine and advised that she take 10 units of insulin two or three times a day. Three days before admission, her friends noted a distinct change from her usual amiable alcoholic self. Her speech became thick and unintelligible. She was confused, often failed to recognize her friends, and was given to fits of choleric temper which were succeeded by periods of apathy. During one of these periods, she asked her lover to give her an injection of insulin. Since she was obviously ill, he decided to give a double dose, to make certain that "at least some of it would work." She immediately lapsed into coma, which her friends assumed was alcoholic. She remained in that state for 36 hours. At the end of that time, she began to have convulsions, and it was decided to call an ambulance.

On admission, she was in deep coma with a double Babinski toe sign. Physical examination and complete laboratory workup were otherwise entirely normal, except that the blood sugar was reported as below 20 mgms. per cent. The usual routine of glucose injections, vitamins by Levin tube, and transfusion, were instituted; and by the fourth hospital day, she was no longer in coma. At this time, she presented a very bizarre picture. Vocalization consisted of one or two meaningless words, buried in a stream of unintelligible jabber. Otherwise she gave no evidence of any contact with her environment and would frequently assume catatonic postures. When stimulated, and frequently without stimulation, she showed evi-

dences of diffuse hypothalamic sympathetic discharge. Her palpebral fissures widened, exophthalmos occurred, and the pupils dilated so that the iris was no longer visible. The pulse rate increased from 100 to 140, the systolic blood pressure from 120 to 170; she would strike out blindly, in the direction of any stimulation, and would literally snarl and spit. Yet, if left alone, she was often quiet and even docile. The reader will, of course, recognize the similarity between this picture and that seen in the decorticate animals described by Bard[13] under the term "mock rage." Obviously in this case, the cortex had been subjected to hypoglycemia for so long a period that irreversible changes had set in; and it was too late for the giving of glucose to reverse the process completely. The cortex was seemingly damaged beyond repair; but the lower centers, with their lessened metabolic requirements, had survived.

After being observed for 30 days without further change, C. S. was sent to a State hospital where a diagnosis of catatonic dementia præcox was made. She eventually died of pneumonia; and an autopsy revealed cerebral softening. Histopathological sections have not as yet been completed, but there can be no doubt as to the severe organic damage suffered by the cortex in this case.

Such cases are not uncommon, but prior to insulin therapy, were usually observed on medical services where the psychiatric involvement was usually not stressed.

Symptoms Due to Oxygen Lack

The respiration of the brain is unusual in at least one other major point. Not only is it essentially restricted to carbohydrate as a substrate, but its quantitative respiration is very intense.[14] For example, the oxygen consumption per unit mass is about thirty times as rapid for gray matter as for muscle or peripheral nerve. In addition, the human brain is exceedingly sensitive to oxygen lack. Indeed, as in the case of glycogen, mentioned before, it is highly probable that no great oxidizing reserve is present in the cells. The oxygen which is present in the capillary bed lasts for 10 seconds; and glycolysis, which begins immediately, does not long supply the needed energy.[15]

The survival time of nervous tissue following oxygen lack can be determined most accurately by measuring the loss of electric potentials or the failure of synaptic transmission. Though transmission through the superior cervical ganglion can resist almost an hour of anoxia, neurons in the central nervous system become electrically quiescent in a few seconds.[16] Characteristic survival times are: motor cortex, 15 seconds; corona radiata, 20 seconds; optic thalamus (geniculate body), from 15 to 30 seconds; and cerebellum, 10 seconds. In the medulla, bursts continue for 50 seconds. Note how neatly this scheme falls in with that previously outlined for insulin hypoglycemia.

Thus, hypoglycemia is brought into peculiarly close relationship with hypoxemia, for if either substrate or oxygen is deficient, metabolism must be interfered with. Indeed, the prediction has been confirmed in man,[17] the dog[18] and the mouse[19] that a lowered supply of dextrose to the brain decreases its oxygen consumption. The synergic effects of a decrease in dextrose and in oxygen described by Gellhorn[20] require more elaborate discussion than time permits, but his results may be summarized as follows:

1. Hypoglycemia acts on the central nervous system in a way similar to oxygen deficiency. In both instances, the rate of oxidation is decreased.

2. The sensitivity of the central nervous system to oxygen deficiency is greatly increased in hypoglycemia.

3. The combination of hypoglycemia plus oxygen deficiency induced by insulin and the inhalation of an oxygen deficient gas mixture produces a more effective stimulation of the sympathetic nervous system, as measured by the blood pressure response, than even the inhalation of pure nitrogen.

It thus appears that some effects of hypoglycemia, at least, are related to anoxia, and, conversely, that anoxia acts by interfering with dextrose metabolism.

In addition, the most significant effects of low oxygen tension or low blood sugar are on the higher centers of the cortex. If any normal person is subjected to a sufficient degree of oxygen want or hypoglycemia, there is an insidious and progressive loss of reasoning, judgment and insight.

Finally, the reaction of the sympathetico-adrenal system when there is want of oxygen or glucose offers a striking example of the prompt and automatic adjustments which preserve the constancy of the internal environment. Cannon[21] has observed that sympathectomized animals faint or collapse in a short while when exposed to low oxygen tensions, and McFarland[22] has noted that neurotic patients with clinical signs of sympathico-adrenal disturbance also appear to be unusually sensitive to lack of oxygen. Similarly in hypoglycemia, there is an increased discharge of adrenalin in an attempt to maintain homeostasis.

Clinical Symptoms of Oxygen Lack

Tremors of eyelids, fingers, and other parts of the body are observed during sudden exposure to anoxia simulating 16,000 to 18,000 feet altitude.[2] With more extreme oxygen lack, ataxia, muscular rigidity and tonic-clonic cramps are commonly observed. One of McFarland's subjects, for example, while inhaling 8.5 per cent oxygen, was unable to make the movements necessary to complete a simple form-board test. His eyes were fixated on the blocks, but his movements were jerky and his fingers were too rigid to return the blocks to their proper places. He knew where they should be placed, but his motor reactions were too impaired to carry out the task. The symptoms disappeared completely when he inhaled oxygen.[22]

It has also been noted that in unacclimated subjects tested in low oxygen chambers at sea level, significant differences in memory occur. Studies of this function show that the average college student at sea level can remember eight to nine out of 10 paired words. While inhaling oxygen percentages simulating 10,000 to 12,000 feet, he is impaired approximately 10 to 15 per cent.[23] The average college professor is often excelled by his own students at a test of this type. This suggests that the loss of memory for immediate recall, which is a familiar part of the aging process, may be related to anoxia. Interestingly, Cameron and his associates[24] have presented evidence indicating that in individuals suffering from psychoses of the senium there is a diminished oxygen consumption by the cerebral tissues. Obviously, variations in memory may be

related to many complex and unexplored factors, but the suggestion that they are due to changes in oxidation and circulation at least offers some hope of a therapeutic approach to the problem.

The effects of prolonged exposure to oxygen lack were first described by Monge.[25] While much had been written on the experimental side, Monge was the first to establish the existence of a definite clinical entity produced by maladaptation to life at high altitudes. His work among the mountaineers is really a classic of clinical observation, although his neurological data are exceedingly meager. His psychiatric observations, on the other hand, are very keen, although he himself was not a psychiatrist. He noted disturbances of memory and behavior "in which the whole psychic apparatus seemed to be altered." He quotes an engineer who during such attacks would make gross mistakes in drawing and arithmetic which he would immediately note on returning to a lower altitude. As the disease advanced, the patients would become apathetic and indifferent to their environment. Despite the fact that these patients soon learned that the descent to sea level would effect a cure, they frequently did not have the initiative to suggest such a change. As a matter of fact, prior to the recognition of this syndrome, most of the patients were considered neurotic or malingerers; for when they reached sea level to consult a physician, their symptoms had, of course, disappeared.

Barach and Kagan,[26] working at Bellevue, also concluded that variations in the oxygen concentration of inspired air beyond that to which the patient is accustomed, result in marked changes in mental functioning. The disturbance was noted both in normals and psychoneurotic patients exposed to inhalations of low oxygen atmospheres. The normals subjected to low oxygen atmospheres revealed marked changes in affective behavior with impairment of emotional control. In 59 per cent, elation and flightiness terminated in lethargy; and in 41 per cent, mental dullness was present from the outset. The "retention and recall" test in 15 of the 17 normals (students) showed a larger incidence of errors and impaired memory after exposure.

The patients (eight cases of anxiety neurosis and one of depersonalization) after inhalation of the low oxygen mixture, revealed an even more marked lack of emotional restraint, with feelings of

exaggerated self-esteem and sexual preoccupations. Their moods ultimately changed from a type resembling a hypomanic state to dullness and lethargy. Patients who did not show elation were dull and lethargic from the beginning. Interestingly, many of the patients made fewer errors than usual in the "retention and recall" test during the period of hypoxia. The authors suggest that the low oxygen atmosphere may have released these persons from their usual preoccupations, and, in thereby relaxing them, made for a superficially better intellectual performance. However, with respect to insight, judgment and emotional content, much more marked impairment was present than in the student group. Most of the patients expressed the suspicion that some stimulating drug or ether had been added to the gas mixture, and all expressed a fear of death by suffocation.

Finally, the authors noted that the inhalation of high oxygen atmospheres by patients with previously existing anoxia also produced marked disturbances in mental functioning. Irrationality, delirium and stupor frequently followed the initial exposure; but when these patients became acclimated, the mental disturbances disappeared and were frequently followed by the appearance of cheerful and optimistic mental states.

In view of these results, it might be of some interest to relate a personal experience with oxygen lack. About five years ago, while serving as ship surgeon, the writer made the acquaintance of an army officer stationed with the British military forces at Jamaica. When our boat landed, I was invited to accompany him to the barracks for lunch. The Newcastle military barracks are about 4,000 feet above sea level, and we made the ascent very rapidly by fast automobile. On route, I felt very gay and happy, but this feeling soon gave way to headache and mild nausea. At the luncheon, which included several alcoholic drinks, I on several occasions forgot my friend's rank which I knew perfectly well. As I arose from the table, I noted that my legs were wobbly and my hands somewhat tremulous. After lunch, we visited the garden. I could not remember, or had specific anomia for, certain of the better-known flowers. My friend had developed a certain enthusiasm for chess, a game which I had taught him on board ship. He beat me rather easily, despite the fact that he was an absolute novice, and my game

at least respectable. I not only found it difficult to concentrate, but I actually made many foolish mistakes. It seemed much more important to finish the game than it did to win. This was not my usual procedure, for at that time I took my chess rather seriously. Reference was made to a deck-tennis game which I had lost the day before. I remarked with some irritation that I had lost on an unfair technicality. Yet, the day before, I had accepted my loss with grace and had quickly repressed my annoyance with a lineman's decision which had cost me the game.

As soon as I returned to sea level, these symptoms immediately disappeared. It would not be too difficult to analyze many of my own personal characteristics in that particular experience, but the thing of importance for the present discussion is the fact that under an atmosphere of diminished oxygen tension, my thinking and attitudes changed, and that when I returned to a more normal environment for myself, my behavior immediately reverted to "normal."

I later discussed the incident with my host who insisted that it was a not unusual occurrence in unacclimated persons.

It should of course be stressed that the anoxia in this case was really a summation of atmospheric and histotoxic (alcoholic) anoxia, and that either one of itself would not have been sufficient to produce the marked distortion in behavior.

The various neuropsychiatric disorders which follow carbon monoxide poisoning (which replaces the oxygen in the oxyhemoglobin) drug poisonings (which frequently directly depress brain oxygenation) and alcohol (which produces a histotoxic anoxia) will serve as sufficient examples of well-known clinical entities which interfere with brain functioning by reducing total brain oxidation. Here, too, a phylogenetic sensitivity is noted, and the syndromes vary from those which are completely reversible, through minor cortical involvements, to medullary involvement with death. Obviously, the final clinical picture must vary with the personality of the individual, and coordinated studies of this type have been sadly neglected.

Finally, a word regarding the effects of anoxia on the amelioration of certain of the schizophrenic syndromes. It is now generally accepted that the various forms of therapy that are successful have

in common the fact that they depress cerebral metabolism. Sakel, who deserves credit for one of the great advances in modern psychiatry, concocted a theoretic interpretation which Stanley Cobb has justly criticized.

Lest we be similarly accused in the future, it is important that we recognize that treatments designed to depress cerebral metabolism or to induce hypoxia or anoxia have no rationale in the treatment of schizophrenia, unless the observable results are related to some of the many side effects (sympathetic stimulation, etc.) of this treatment.

Symptoms Due to Vitamin Lack

Our present knowledge indicates that at least three of the vitamins already isolated, are concerned with the proper metabolism of carbohydrate. These are:

1. *Thiamin Hydrochloride.* This substance or more particularly its diphosphoric ester (thiamin pyrophosphate) is concerned in the proper catabolism of carbohydrate at the pyruvic acid stage.[27] In its absence, the breakdown of carbohydrate is interfered with at the pyruvic acid stage, and this substance accumulates in the body fluids.[28] Furthermore, the writer and an associate have shown that in clinical cases of thiamin deficiency, pyruvic acid does accumulate in the blood and spinal fluid.[29, 30]

2. *Nicotinamide.* Nicotinic acid is a constituent of coenzyme 1 or cozymase[31, 32] which is a catalyzer in certain oxido-reductions in the intermediary metabolism of carbohydrate.

3. *Riboflavin.* Riboflavin is a structural element of Warburg's yellow enzyme.[33, 34, 35] The latter is capable of oxidizing a series of metabolic products (hexosephosphate, citrate, malate) by transporting hydrogen to suitable acceptors. It can also transport the molecular oxygen of the air directly to those substances. Furthermore, both cozymase and the yellow enzyme are catalyzers in the synthesis of co-carboxylase from thiamin.[27, 36]

Since, therefore, thiamin, nicotinic acid and riboflavin are all substances of great importance in carbohydrate catabolism, it is perfectly conceivable that a deficiency in any one of them may interfere with proper brain functioning by interfering with the proper utilization of dextrose, the essential foodstuff of the brain.

As a matter of fact, clinical syndromes associated with a deficiency of two of these substances have already been described (see following). Yet many other vitamins are concerned in brain tissue metabolism, and Page's textbook on brain chemistry[37] in 1937 had already noted that 21 enzymes were to be found in the brain. For the present, however, we must confine ourselves to the symptoms resulting from lack of thiamin and nicotinic acid.

A. SYMPTOMS RESULTING FROM THIAMIN DEFICIENCY

1. *Neurasthenic Syndrome*

Tension and irritable weakness states, frequently alluded to as neurasthenia and nervous exhaustion, are manifested in complaints of fatigability, weakness and exhaustibility, head pressures, poor sleep, irritability, feeling of tenseness, inappetence, various aches and pains, subjectively poor memory, and difficulty with concentration. Additional complaints referable to the bowels, heart, skin, and genito-urinary apparatus are not uncommon. The etiology of this condition is not clear, but it is generally thought to be one of the more constitutionally determined of the minor psychiatric reaction types. Various etiologies have been suggested, but none satisfactorily explains the picture. Freud himself felt that neurasthenia was the result of current physical factors and suggested that it was the direct physical result of excessive masturbation.[38] As various hormones were isolated, each in turn was held responsible. In this vitamin era, similar indictments are being made.

With a thiamin-poor diet, Jolliffe and his coworkers[39] were able to produce a neurasthenic syndrome in four out of five experimental subjects. Their subjects (internes) complained of fatigue, lassitude, anorexia, precordial pain, burning of the feet, dyspnea on exertion, muscle cramps and palpitation. The objective signs observed were skin hyperesthesia in a sock distribution, changes in the electrocardiogram, and calf muscle tenderness. Symptoms were observed as early as the fourth day, and objective signs as early as the fifth, although one subject developed no definite symptoms or objective signs in 30 days with a diet estimated to contain only 62 per cent of his thiamin requirement.

The addition of thiamin alone to the experimental diet caused all symptoms to disappear within three days, and the objective signs within six days.

It is interesting that McLester[40] had previously noted that many of the pellagrins seen at the Hillman Hospital in Birmingham were considered neurasthenics before the objective signs of pellagra became manifest. It is now well recognized that few cases of pellagra depend exclusively on a deficiency of nicotinic acid and that there is an additional deficiency of thiamin in most such cases. He suggested that many of the nervous and mental symptoms of pellagra depend primarily on a lack of sufficient thiamin in the dietary regimen.

In 1940, the carefully-controlled study of Williams and his coworkers[41] apparently established the fact that a syndrome resembling neurasthenia could be produced by a diet deficient in thiamin. Their otherwise excellent study suffers from the fact that they utilized as experimental subjects five formerly psychotic individuals (three cases of dementia præcox, one of psychosis with poliomyelitis, and one of paranoid psychosis) and one psychoneurotic. Since somatic complaints of the type listed in neurasthenia are very common in such patients, the results are open to serious criticism. The problem is additionally complicated by the fact that similar syndromes have been reported as cured by nicotinic acid,[42] pyridoxine[43] and vitamin E.[44] Furthermore, it should be kept in mind that the clinical picture of this syndrome is extremely varied, that many such pictures occur in apparently well-nourished individuals, and that these symptoms frequently respond to psychotherapy.

It should therefore not be inferred that all neurasthenia is based on thiamin or other nutritional deficiency. It does, however, seem fairly certain that a syndrome possessing many of the characteristics of the ill-defined neurasthenic syndrome can be produced by nutritional deficiency. The need to isolate this group from neurasthenic syndromes of other etiology is emphasized.

2. *Cortical Dysfunction Associated with Acute Peripheral Neuropathy*

Acute peripheral neuropathy in the alcohol addict is now generally accepted to be the result of nutritional deficiency and more particularly to be related to a deficiency in thiamin hydrochloride. It is therefore pertinent that the writer has never seen a case of acute peripheral neuropathy that did not simultaneously show some evidence of cortical dysfunction, although the type of organic psychopathy varies.[45] This problem is still under investigation, but it is already fairly obvious that many of these syndromes are related to a nutritional deficiency. Finally, the frequent association of the Korsakoff syndrome and the almost invariable association of the Wernicke syndrome with polyneuropathy of nutritional origin, suggests a further link.

3. *Wernicke's Syndrome*

In 1881, Karl Wernicke,[46] on the basis of three cases studied carefully during life and at autopsy, delineated a clinical syndrome characterized by clouding of consciousness, varying ophthalmoplegias and ataxia. The author did not indict alcohol as the causative agent, but suggested that various toxins, including alcohol, might produce the clinicopathologic picture. Nonetheless, most subsequent cases were reported in inebriates, and alcohol gradually came to be accepted as the etiological basis of this condition. As a matter of fact, Wernicke's original case did not occur in an alcoholic.

The patient was a 20-year-old seamstress who was admitted to the Charité following a suicidal attempt with sulphuric acid. She left the hospital after several days, but soon thereafter persistent and intractable vomiting set in, probably as a result of pyloric stenosis. The vomiting continued; and after one month the young woman became stuporous and developed ophthalmoplegia and ataxia. In addition, there was moderate swelling of the optic discs with associated retinal hemorrhages. Her condition gradually became worse, and she died one week after the onset of this complication. Wernicke's other two cases, however, did occur in alcoholics who were admitted in delirium. The essential pathology was described by Wernicke and has been further elaborated by others. In

general, the lesions are confined to the periventricular gray matter about the third ventricle and hypothalamic region. They are characterized by small foci of degeneration and varicose deformities of the blood vessels. There is subacute necrosis of the adjoining parenchyma, and small petechial hemorrhages are frequently but not always found throughout the lesions.

In addition to innumerable reported cases in inebriates, a review of the literature reveals some 40 cases described in non-alcoholic individuals. These latter are usually associated with gastro-intestinal disorders or carcinomata accompanied by cachexia and vomiting. Many of the authors suggested a metabolic etiology, but opinion regarding the nutritional origin of the Wernicke syndrome was not crystallized until 1938, when Alexander and his co-workers[47] were able to produce it in pigeons fed on a thiamin-deficient diet. They could not reproduce this disease in pigeons fed thiamin, even though they were deprived of all other vitamins or of any one other vitamin for a period of over six months. In 1940, Alexander[48] amplified his original report and showed conclusively that the lesions of Wernicke's polioencephalopathy occurring in man, and the disease which he produced experimentally in vitamin B_1 deficient pigeons were identical in their topographical distribution, and in their morphological and histological characteristics.

The writer and associates,[49] have recently reported their clinical findings in 27 cases. Of the total, three occurred in non-alcoholics (two depressed patients who refused to eat, and one case of pulmonary tuberculosis with associated vomiting). The other 24 occurred in chronic alcoholics. The results may be summarized as follows:

1. The syndrome as originally described by Wernicke is probably a combination of several nutritional deficiencies affecting the nervous system and need not necessarily be complete in any case.

2. The results obtained indicate that: (a) The ophthalmoplegia is a thiamin deficiency. (b) The clouding of consciousness may be related to anything which interferes with proper brain metabolism. Among the known offenders are lack of carbohydrate, lack of oxygen, lack of thiamin, nicotinic acid and riboflavin, and probably

lack of many other substances now under investigation. (c) The ataxia is difficult to evaluate, and its response to therapy has not as yet been worked out.

3. Other deficiency syndromes (pellagra, nicotinic acid deficiency encephalopathy, riboflavin deficiency) may and do superimpose themselves on the more usual Wernicke picture; and these require specific treatment.

4. The ophthalmoplegia is invariably preceded or accompanied by peripheral neuropathy. Since the latter is associated with a thiamin deficiency, this finding tends to confirm Alexander's thesis that they have a common etiology and that the polioencephalopathic changes represent a more complete deficiency in thiamin.

5. Delirium, with its marked increase in psychomotor activity, and hence in total metabolism, usually precedes the development of this syndrome. In this type of case, the early administration of thiamin will prevent the development of ophthalmoplegia.

6. All patients who received adequate vitamin therapy recovered. In the recovered cases, the development of a Korsakoff syndrome is the rule. The latter does not show a consistent response to thiamin therapy, as has frequently been asserted.

B. SYMPTOMS RESULTING FROM A DEFICIENCY OF NICOTINIC ACID

Pellagra was first described by a Spanish physician, Gaspar Casal, in 1735, but his observations were not published until 1762, three years after his death. The first published report appeared in the French literature in 1755, but the author, formerly physician to the French ambassador at the Spanish court, freely acknowledges his debt to Casal.[50] States of mania, depression, and confusion are described as accompanying the *mal de la rosa,* and their severity and chronicity are stressed. "Without doubt these are produced by metastasis to the brain of the acrid and malign humors which produced this malady." He cites the example of a woman who, "during one of the melancholy deliriums so frequent in this disease, had a great desire to feed herself from cow's butter, for which she spent all her property, and she was cured."

Almost immediately following the discovery of the value of nicotinic acid in the treatment of certain manifestations of human pellagra, a long series of articles[51, 52, 53, 54] appeared concerning its use-

fulness in the treatment of the cerebral disorders seen in pellagrins. Spies and his coworkers[51] state that pellagrins are noted for the multiplicity of their complaints, among which are many that are usually classified as neurasthenic. The most common of these symptoms are fatigue, insomnia, anorexia, vertigo, burning sensations in various parts of the body, numbness, palpitation, nervousness, a feeling of unrest and anxiety, headache, forgetfulness, apprehension, and distractability. The conduct of the pellagrin is normal, but he feels incapable of physical or mental effort, even if he is ambulatory. Spies and coworkers noted "neurotic" symptoms showed a prompt response to nicotinic acid therapy. Probably of greater significance than the relief of "neurotic" symptoms following nicotinic acid, is their observation that these symptoms return when, without the patient's knowledge, nicotinic acid is withdrawn and another medicament of similar appearance is substituted for it.

The more obvious mental manifestations of pellagra are the various organic psychoses which complete the diagnostic triad of diarrhea, dermatitis, and dementia. The most common is perhaps that in which loss of memory, disorientation, confusion, and confabulation are present. There are also types in which excitement, depression, mania and delirium may occur. In experience at Bellevue Hospital, a paranoid condition is common in pellagrins, as in many other organic psychiatric pictures. Spies and his associates report that all their treated psychotic patients recovered, but the psychosis in most of their cases was only of one to two weeks' duration. These findings can be confirmed by experiences at Bellevue. The writer would, however, emphasize the fact that careful psychiatric examination reveals that these patients are frequently left with residual organic memory defects. In the psychoses of longer duration associated with pellagra, the response to nicotinic acid is certainly not spectacular, and specific therapy may not help at all. This does not mean that a lack of nicotinic acid was not important in the genesis of the mental picture. It does, however, stress the fact that metabolic disturbances finally proceed to structural changes. When this latter stage is reached, the process may become irreversible. Hence therapy to be efficacious should be instituted before structural changes occur. It must similarly be

stressed that many of the acute excitements and deliria associated with pellagra may clear without nicotinic acid therapy. Finally, pellagrins are usually lacking in other factors which are contained in the well-balanced diet and which are probably necessary for normal brain metabolism. It is therefore suggested that adequate amounts of other vitamins be given to pellagrins with encephalopathic manifestations, in order to insure maximal therapeutic results.

Cleckley, Sydenstricker and Geeslin[55] have reported on 19 stuporous patients who showed a remarkable response to nicotinic acid therapy. They concluded that hebetude grading into profound stupor may be the only sign of severe acute pellagra, and that therapeutic trial of nicotinic acid is justifiable as the only method at present available for the diagnosis of such cases.

Jolliffe, Bowman, Rosenblum and Fein[56] have reported 150 cases of an "encephalopathic syndrome," heretofore almost invariably fatal, which they believe is caused by nicotinic acid deficiency. This syndrome may occur as the only manifestation of a deficiency disease or it may occur in association with pellagra, polyneuritis due to vitamin B_1 deficiency, or the ophthalmoplegia associated with Wernicke's disease. The clinical picture of this syndrome is more or less well defined and is characterized by clouding of consciousness, cog-wheel rigidities of the extremities, and uncontrollable sucking and grasping reflexes. To be excluded are the encephalopathic manifestations of groping, grasping, and sucking which may occur during the course of delirium tremens, infectious diseases with delirium, expanding intracranial lesions, advanced cerebral arteriosclerosis, and other diseases. Jolliffe also notes that some of the cases previously described by Spies, Matthews, Sydenstricker, and Cleckley, and their coworkers, may have belonged to this group.

Since not all of the cases showing this syndrome presented the usual skin and mouth lesions associated with pellagra, it was assumed that this syndrome represents an acute complete nicotinic acid deficiency which develops so rapidly that the structural changes in the skin and mouth, characteristic of pellagra, do not have time to occur. Patients manifesting this syndrome treated by hydration plus thiamin hydrochloride almost invariably die (95

per cent). Patients treated by hydration plus concentrates rich in the vitamin B-complex show a moderate drop in mortality (50 per cent); but when these patients are treated by hydration plus nicotinic acid a marked drop in mortality occurs (15 per cent).

Finally, a word about two other syndromes which have frequently been associated with vitamin deficiency, but in which the evidence, in the writer's opinion at any rate, is still inconclusive.

Delirium Tremens

The literature contains many articles indicating that thiamin chloride[57, 58, 59] and nicotinic acid[60, 61, 62, 63] are either specific or have considerable value in the treatment of delirium tremens. On the other hand, Spies and his coworkers[64] reported negative results with nicotinic acid, and Rosenbaum[65] noted no essential difference whether vitamin B_1 or nicotinic acid be given or omitted in the routine therapy of these cases. As a matter of fact, he reported the experimental production of an attack of delirium tremens in a chronic alcoholic who received huge doses of thiamin chloride and nicotinic acid, but was allowed to drink about one quart of whiskey a day. The delirium began 13 days after this regimen was started.

Published results of the writer and coworkers,[66, 67, 68] indicate clearly that deficiency of thiamin and nicotinic acid is not specific in the causation of delirium tremens and that administration of these vitamins is not specific in the treatment of this disease. They do, however, believe that delirium tremens is a factor of considerable importance in producing other nutritional disturbances of the nervous system (Wernicke's syndrome, nicotinic acid deficiency encephalopathy, peripheral neuropathy). The marked increase in psychomotor activity which accompanies the delirious states so raises the metabolic requirements that clinically latent deficiency states may become manifest. The writer and coworkers have, however, seen too many cases of delirium tremens recover entirely on saline and fluids to include nicotinic acid or thiamin in the specific treatment of this condition. They believe, however, that thiamin and nicotinic acid, as well as the entire B-complex, should be given to all patients with delirium in order to prevent the development of peripheral neuropathy, Wernicke's syndrome, and nicotinic acid deficiency encephalopathy, and perhaps to prevent the development

of as yet unknown types of encephalopathy related to nutritional disturbances. Delirium tremens may, of course, be related to some as yet undiscovered or untried vitamin, but the evidence for thiamin and nicotinic acid is very meager indeed.

Korsakoff's Syndrome

This psychosis consists in deficient power of retention for recent events, with a tendency to confabulate, and disorientation for time, place and person. It may or may not be associated with peripheral neuropathy. Although most usually associated with chronic alcoholism, it is seen in conjunction with many other conditions (head injury, diabetes, arteriosclerosis, subarachnoid bleeding, toxic and drug psychosis, etc.). Because of the frequent association of this psychosis with peripheral neuropathy, lack of thiamin has long been suspect, and several reports tending to confirm this[69, 70] have already appeared. These reports are probably the result of premature optimism. Experience at Bellevue with the syndrome convinces the writer that not all cases of this type are the result of nutritional disturbances, and that even where such disturbances do exist, the rôle of thiamin is still to be determined. The writer[71] has previously reported that the confabulatory features of an acute delirium usually clear while the patient is receiving a vitamin B deficient diet. In the more chronic cases, Bowman and his associates[72] have shown that thiamin seems to help in the recovery of these patients, but is in nowise specific. In these latter cases, it may be argued that the changes have become irreversible. At any rate, the rôle of thiamin in the genesis and treatment of the Korsakoff syndrome still remains to be determined, and the evidence at hand is not at all convincing.

The facts presented indicate clearly a need for psychiatrists to reorient themselves and to become conversant with certain facts regarding the nutritional aspects of brain function. With rare exceptions, one is impressed with the lack of interest in the psychological aspects of the various reported illnesses—a defect which the psychiatrist, properly trained or working in conjunction with a properly trained personnel, could remedy.

It would, therefore, seem logical for the psychiatrist, as a student of nervous economy, to evidence interest not only in dynamic psychiatry, but also in the dynamic physiology and metabolism of the brain, and more particularly in the interrelationship of the two.

Medical Service, Psychiatric Division
Bellevue Hospital
New York, N. Y.
Department of Psychiatry
New York University College of Medicine
New York, N. Y.

REFERENCES

1. Himwich, H. E., and Nahum, L. H.: Am. J. Physiol, 90:389, 1930.
2. McFarland, R. A.: Am. J. Psychiat., 97:858, 1941.
3. Jolliffe, N.: Trans. Assn. for Research in Nerv. and Ment. Dis., 19:144, 1939.
4. Sperry, W. M., Waelsch, H., and Stoyanoff, V. A.: J. Biol. Chem., 135:281, 1940.
5. Rose, W. C.: The Harvey Lectures, 30:49, 1936.
6. Jervis, G. A.: Arch. Neurol. and Psychiat., 38:944, 1937.
7. Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J. F.: J. N. M. D., 89-273, 1939.
8. Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J. F.: J. A. M. A., 112-1572, 1939.
9. Himwich, H. E., and Nahum, L. H.: Am. J. Physiol., 90:384, 1929.
10. Himwich, H. E., Frostig, J. P., Fazekas, J. F., and Hadidian, Z.: Am. J. Psychiat., 96:371, 1939.
11. Hoagland, H., Himwich, H. E., Campbell, E., Fazekas, J. F., and Hadidian, Z.: J. Neurophysiol., 2:276, 1939.
12. Baker, A. P.: Am. J. Psychiat., 96:109, 1939.
13. Bard, P.: Am. J. Physiol., 84:490, 1928.
14. Gerard, R. W.: Arch. Neurol. and Psychiat., 40:985, 1939.
15. Kerr, S. E., and Ghantus, M. L.: J. Biol. Chem., 117:217, 1937.
16. Bronk, D. W., and Larrabee, M. G.: Am. J. Physiol., 119:279, 1937.
17. Damashek, W., and Myerson, A.: Arch. Neurol. and Psychiat., 33:1, 1935.
18. Himwich, H. E., Bowman, K. M., Wortis, J., and Fazekas, J. F.: Science, 86-271, 1937.
19. Holmes, E. G.: Biochem. J., 24:914, 1930.
20. Gellhorn, E.: J. A. M. A., 110:1433, 1938.
21. Cannon, W. B.: The Wisdom of the Body. W. W. Norton and Company, New York, 1932.
22. McFarland, R. A., and Barach, A. L.: Am. J. Psychiat., 93:1315, 1937.
23. McFarland, R. A.: Bureau of Air Commerce, Department of Commerce, Report No. 13, May, 1938.
24. Cameron, E., Himwich, H. E., Rosen, S. R., and Fazekas, J. F.: Am. J. Psychiat., 97-566, 1940.
25. Monge, C.: Arch. Int. Med., 59:32, 1937.
26. Barach, A. L., and Kagan, J.: Psychosomatic Med., 2:53, 1940.
27. Banga, I. L., Ochoa, S., and Peters, R. A.: Biochem. J., 33:1109, 1939.
28. Lu, G. D.: Biochem. J., 33:774, 1939.
29. Bueding, E., and Wortis, H.: Proc. Soc. Exp. Biol. and Med., 44:245, 1940.

30. Wortis, H., and Bueding, E.: Trans. Am. Neurol. Assn., 66:90, 1940.
31. Warburg, O., and Christian, W.: Biochem. Ztschr., 287:291, 1936.
32. Euler, H. V., Albers, H., and Schienk, F.: Ztschr. Phys. Chem., 240:113, 1936.
33. Warburg, O., and Christian, W.: Biochem. Ztschr., 254:438, 1932.
34. Gyorgy, P., Kuhn, R., and Wagner Jauregg, T.: Naturw., 21:560, 1933.
35. Ochoa, S., and Rossiter, R. G.: Biochem. J., 33:2008, 1939.
36. Lipton, M. A., and Elvehjem, C. A.: Cold Springs Harbor Symposium on Quantitative Biology, 7:184, 1939.
37. Page, I. H.: Chemistry of the Brain. C. C. Thomas, Springfield, Ill., 1937.
38. Freud, S.: Collected Papers, Vol. 1, p. 226, Int. Psychoanalytic Press, 1924.
39. Jolliffe, N., Goodhart, R., Gennis, J., and Cline, J. K.: Am. J. Med. Sci., 198:198, 1939.
40. McLester, J. S.: J. A. M. A., 112-2110, 1939.
41. Williams, R. D., Mason, H. L., Wilder, R. M., and Smith, B. F.: Arch Int. Med., 66:785, 1940.
42. Frostig, J. P., and Spies, T. D.: Am. J. Med. Sci., 199:268, 1940.
43. Spies, T. D., Bean, W. B., and Ashe, W. F.: J. A. M. A., 112-2414, 1939.
44. Spies, T. D., and Vilter, R. W.: Southern Med. J., 33:663, 1940.
45. Wortis, H., and Jolliffe, N.: New York State J. of Med., (in press).
46. Wernicke, K.: Lehrbuch der Gehirnkrankheiten 1881, Kassel, Vol. II, p. 229.
47. Alexander, L., Pijoan, M., and Myerson, A.: Trans. Am. Neurol. Assn., 64:135, 1938.
48. Alexander, L.: Am. J. Pathol., 16:61, 1940.
49. Jolliffe, N., Wortis, H., and Fein, H. D.: Arch. Neurol. and Psychiat., 45:560, 1941 (abstract).
50. Casal, G.: Quoted by Major, R. H.: Classic Descriptions of Disease. C. C. Thomas, Springfield, Ill., 1932.
51. Spies, T. D., Aring, C. D., Gelperin, J., and Bean, W. B.: Am. J. Med. Sci., 196:461, 1938.
52. Matthews, R. S.: J. A. M. A., 111:1148, 1938.
53. Sydenstricker, V. P., Schmidt, H. L., Fulton, M. C., New, J. S., and Geeslin, L. E.: Southern Med. J., 31:1155, 1938.
54. Spies, T. D., Grant, J. M., Stone, R. E., and McLester, J. B.: Southern Med. J., 31:1231, 1938.
55. Cleckley, H. M., Sydenstricker, V. P., and Geeslin, L. E.: J. A. M. A., 112:2107, 1939.
56. Jolliffe, N., Bowman, K. M., Rosenblum, L. A., and Fein, H. D.: J. A. M. A., 114:307, 1940.
57. Kiene, H. E., Streitweiser, R., and Miller, H.: J. A. M. A., 114:2191, 1940.
58. Kloster, J.: Nervenarzt, 2:413, 1938.
59. Santesson, G.: Svenska Lakartidn, 36:926, 1939.
60. Pagniez, P.: Pr. Med., 47:1682, 1939.
61. Mainzer, F., and Krause, M.: British Med. J., 2:331, 1939.
62. May, A. M.: Med. Rec., 150:124, 1939.
63. Sydenstricker, V. P.: Paper presented at annual meeting of Amer. Psych. Assn., Cincinnati, Ohio, May, 1940.
64. Spies, T. D., Aring, C. D., Gelperin, J., and Bean, W. B.: Am. J. Med. Sci., 196:461, 1938.
65. Rosenbaum, M., Piker, P., and Lederer, H.: Paper presented at annual meeting of Am. Psych. Assn., Cincinnati, Ohio, May, 1940.
66. Wortis, H., Wortis, S. B., Frank, P., Joliffe, N., and Bowman, K. M.: Arch. Neurol. and Psychiat., 41:655, 1939.
67. Bowman, K. M., Wortis, H., and Keiser, S.: J. A. M. A., 112:1217, 1939.
68. Wortis, H.: Quart. J. Stud. Alcohol, 1:251, 1940.
69. Weiss, S.: Cecil's Textbook of Medicine. W. B. Saunders and Company, IV, p. 562. Ed. 1937.
70. Friedman, A.: Schweiz. Arch. Neurol. and Psychiat., 39:263, 1937.
71. Wortis, H.: New York State J. Med., 29:1178, 1939.
72. Bowman, K. M., Goodhart, R. S., and Jolliffe, N.: J. N. M. D., 90:569, 1939.

THE ORGANIC IN PSYCHOTHERAPY AND PSYCHOGENESIS*

BY GEORGE S. SPRAGUE, M. D.

He who wishes to practice psychotherapy is at once challenged to think through very accurately his own philosophy and convictions concerning the structure of the human mind. He must realize that an honest conviction in the theoretical efficacy of psychotherapy must be premised upon the prior postulation of psychogenesis. In turn, before he can feel assured that he can accept psychogenesis as a fact, he must have settled all misgivings as to whether there are definite causal relationships between the functional and the organic in the workings of the human mind. This paper proposes an inquiry into the relationships which may be established, some of the conclusions which may be drawn therefrom, and some perhaps helpful attitudes which such understandings would enable us to adopt. In preparation for this, it will be necessary to suggest the spirit in which the communication is presented by discussing some definitions of such conceptions as organic, functional, mental, and the like, with which we are to deal.

In psychiatry, as in other fields, there are certain conceptions which are more or less tacitly accepted as everyday facts of experience and which we are apt to assume we understand in about the same way as do our fellows. These include our conceptions of the human mind, its machinery and its activities. It is the writer's opinion that we have become involved with unclear thinking about these things, and that this, to a large extent, has been a matter of terminology. There must be about mental phenomena certain simple concepts which can be said simply, thus enabling us to form new realizations about the subject or to acquire a more comfortable comprehension of our present hypotheses about the activities of the mind and their import in psychotherapy.

This is brought to our attention when we seek to practise the art of psychotherapy. We are likely to become so interested in the results we strive for that we do not stop to question again the conceptions on which our methods, whether wittingly or unwittingly, are premised. The psychotherapeutic pattern is likely to be thought

*Read at the thirteenth annual meeeting of the American Psychopathological Association at Cincinnati, Ohio, May 18-19, 1940.

of in some such terms as these: The therapist presents ideas to the patient. These ideas, by their interaction with those of the patient, cause modification of his ideas; and as a result the patient will somehow modify his thinking, feeling or action. If the psychotherapist is not satisfied with the results, he will then use more of the same kind of treatment. In such a conception, the force of ideas is implicit, and one perhaps does not pause to consider the significance of the organic components. We call the procedure a functional one because we think of the force and influence of the ideas as such, as if the thoughts were entities in their own right.

But if we are to escape the pitfalls of mysticism, it is important to keep firm hold on the fact that actual living matter is the basis for all psychiatric study. The organic we have with us always. It is a spatially existent physical agglomeration which might be spoken of as the home address for various qualities, attributes and experiences. The organic is bodily, real; it has physical properties, dimensions and limits. It is what you have, what you are; it is the machinery itself. As with any smoothly-running equipment, its use should not cause devastating alterations of its own structure—playing a piano leaves that instrument essentially the same apparatus it was before. A piano may play different tunes of differing character, but they make no assertions about the instrument except for implying that the equipment was such as could produce them. It would not follow, however, that it must of necessity actually produce those tunes. Similarly the human architecture, and specifically that of the central nervous system, has certain organic structure defining the limits and enabling the performance of its potential activities.

The "functional" is the activity of the organic structure. It is the carrying into reality of some potentiality of the organic machinery. The functional is what you do with what you have. It is the experience of a change which is natural for that organization. The action of the mind, therefore, may be thought of as being a functional process of the brain, and a functional mental disease is one which occurs without morbid structural changes. We must make a distinction here between the finer structural variations occurring in physiological processes such as metabolism or action currents, and the grosser, more permanent alterations which make

lasting changes in organic structure. It is seen, therefore, that the functional changes are those which do not disrupt the integration or the entity of the organization. In the brain, as in all other tissues, mere change is not in itself the crucial issue. The important fact is the maintenance of a certain relative balance among its component elements. It is this balance whose preservation constitutes the organic background, and whose mobility constitutes its functioning. The life processes of the brain maintain its organic structure with relatively little variation, so that its functional exercise still leaves it essentially with the same structure as before except for the practically undetectable alterations which result from protoplasmic memory-experiences.

We do not think of these mnemonic traces as representing "mind." When, however, we consider the subjective experiencing of a memory, some doubt may arise. And when we debate whether thought content, or the having of such a content, or the capacity for having it, is to be called mind, there is more room for divergent views. If we propose to say that the term "mind" is a term designating the sum total of the functionings which actually occur in a given brain, it will be evident that too much function has been included. There is brain functioning of a kind in the mere reception of an auditory sensation, but this is obviously not a mental phenomenon. Tentatively then, let us think of the mind in a somewhat more restricted sense, not as inclusive of the brain's potentiality for action but as being a collective term for that part of the brain's functioning which deals with meaningfulness. In the analogy of a piano's being played upon, mind as thus described would be comparable not to the sounds elicited, but to the fact that the tune played was, say, the national anthem.

The brain by virtue of its enormous associational tracts has the functional capacity for establishing innumerable linkages among its stored memory traces. This association or grouping of various other items to the one under consideration is the basic fact entailed in the conception of "meaning." It is difficult to picture the notion of mind and of mental workings without including the phenomena of meaning. The real problem of understanding meaning becomes one of evaluating the reasons for the formation of just the particular associational linkages which have occurred in each

individual instance. There seem to be two ways in which the process is determined, the one due to personal experience, and the other due to experiences of the surrounding social group. The kind of associational connections which are common to the people around us are likeliest to become ingrained in us by training and by observation. In this way an orthodoxy of mental associations is built up in the individual. It is of practical convenience that from the same stimulus approximately the same associational trend arises in his mind as arises in the minds of his fellows, for only then can he talk in the same language with them. On the other hand, it is from the more personal and individual experiences that one forms certain of his mental associations which may differ more or less from those of his neighbors, thus giving to his mind its personal character.

The result of these two methods for the formation of associations is that the individual builds up an increasingly solid basis of community with his fellows concerning the meaningfulness of a great number of elements. But at the same time, he also develops various meaningful linkages on the basis of his personal involvements, his likes, dislikes and instinctive-affective pressures. Thus, there is no marked difference in the associated meanings for a chair so that little divergence of view among different people arises over this item. The concept of punishment may, however, have widely differing connotations to various individuals. In such an instance, it would necessarily follow that discussion of this topic by those individuals could not proceed on the basis of a common conception. Such a situation attains practical importance when planning psychotherapy.

Whatever is often enough repeated becomes habitual and so requires little conscious notice. The greater part of meaningfulness becomes thus habituated, and like an automatic act such associational sequences usually find an unquestioning acceptance. We could say that much of our own thought consists in not thinking. For example, we do not daily reexamine our associations about a chair. Let him who doubts this statement note the difficulty of forming a definition for some common word for the first time. Meaning, which starts as a connection of elements such that one implies or subsumes others, is then in a position to extend still further by a

process of substitution. When one has had enough experience with an associated group of elements, we know that less attention may be given to the separate items, as their presence in the grouping comes to be assumed. At this stage of development, such items as are dealt with directly, become representatives or substitutes for the whole group of items on the basis of an established habit pattern.

The next level of development, but an easy step from this one, is perhaps the most important single one in the development of the mental function. Instead of an element representing other elements in the immediate stream of associations with it, a further extension of the chain of associations may cause it to represent an element or group less directly linked to itself. Here we have the phenomenon of something "standing for" something else, and this is the pattern of symbol formation. The former tentative idea of the mind as the sum of the brain's functioning which deals with meaningfulness may now, in the writer's opinion, be narrowed to the following: Mind is the sum of those brain activities in which symbolism occurs. With this conception a blow resulting in the exclamation "Ouch!" would not be termed mental, although pain was experienced. But if the individual used such associative functions as would call up the notion, "He struck me," mental activity would be asserted because word symbols have been evoked by association and are used substitutively.

Because this exercise of the mental function is encouraged by teaching and example and because it is so efficient in time and labor saving, it has become essential to existence, and it is therefore readily understandable that we place great value upon our psychic life. Another cause for its great importance to man lies in the fact that he can manipulate his mental functioning without the difficulties and concessions to the realities of the outer world which non-mediated living would necessarily involve. If he can take words and ideas as substitutes for objective realities, he is in a position through them to attain gratification for his longings which would be available in no other way. There is not only the importance of wish fulfillment to be considered, but also a gratification of the innate longing for power which stems from the infantile phase of omnipotence. From these several factors it appears quite under-

standable that man has not only held his mental functions in high regard but has had a distinctly recognizable tendency to overvalue them, even to glorify them. This trend is evidenced by the majority of individuals, but nowhere more strikingly than by the psychotic to whom thought is delusionally enthroned with exaggerated powers and authority.

In everyday life we do the same thing with our calm unquestioning acceptance of such words as "thought," "mind," "consciousness," and similar notions as if they were representative of actual entities. Just here lies one of the important issues requiring clear consideration about the relationships of brain and mind, of organic and functional, and of genesis from organic or from functional, psychic factors. Consider, for example, the actual facts and the ordinary beliefs we would have about them in such an everyday case as the following:

Suppose two persons to be carrying on a conversation in which they agree upon a plan for a fishing trip they will take next Saturday. Their ideas, their plans are valued as if they were absolute entities in their own right. The fishing trip, which has not yet occurred at all in reality, seems as a thought even more real than does the physical activity of speech production which is involved. They think of their plan as being a "real" plan. They consider that they have "made" it by talking it over, that the dialogue and the sense of agreement have given it legitimate birth, so that it now exists more than did the various different noises which they produced with their talking. When they have separated and the words of the plan are called into their mental content by association, they think of the plan as continuing to have "its existence." They would be impatient if asked to include the activities of their vocal cords and intercostal muscles as being parts of "the conversation" which they will assert was "just an exchange of ideas" to which all else involved appears as irrelevant. Not only they, but an observer as well, or their wives when told of the prospect, will likewise accept the priority of the thoughts over any structural bodily details.

The lay individual will be sure that the fishing trip had its origin in some ideas; that, in other words, it was psychogenetically motivated. He fails, however, to take into account both a direct and

an indirect contribution which has not been psychogenetic. Each fisherman in turn has been stimulated to the production of his own further ideas by nonpsychic stimuli. His stimulus has not been ideational but has been acoustic; he has not been given his friend's thoughts although he thinks he has. On the same basis it can be realized that psychogenesis can never take place from outside one's self, as the afferent system has no means of receiving mental contents. This is the direct factor. Also there is an indirect contributing factor, namely the organic and physiological state of the listener, for in addition to such factors as his learning capacity and the disturbing noise of the wind in his ears, there is to be evaluated in a careful study of the facts his individual conditioned reflex patterns of responsiveness to the words, ideas and attitudes which he experiences.

It may seem pedantic to some to call attention to such obvious and detailed items in the patterns of human relationships, but the writer believes that it is in part due to an unreadiness to go carefully into such details, that an incomplete view of an involved situation exists. When such data are well enough understood, words will be coined which can then act as important short-cuts in language. But while these matters still look so complex, we must talk the long way around.

The statement was made in the foregoing that, strictly speaking, psychogenesis can never take place from outside the subject's organism. This is to say on the one hand that he has no means of coming into direct contact with psychic influences except his own, or otherwise stated, that mental phenomena cannot be transmitted from one individual to another because they are merely brain functionings. On the other hand and for the same reason, what may start as an attempt at psychogenetic psychotherapy on the physician's part will likewise be unable to find a medium of transmission to his patient until it has undergone an alteration into a communicable form. This leads us to the dictum that all therapy originating externally to the patient can have no other than an organic mode of influencing the patient. If the therapy starts as an organic one, as by the giving of drugs or the administration of tube feedings, the process of reception is a direct one. If a psychotherapeutic approach is made, the process might be compared to

the action of an electric rectifier which allows only one type of current to pass through it regardless of what type it receives.

In a strict sense, all our therapeutic efforts for psychiatric patients will have at least to pass through a reducing valve, as it were, in which they become of organic and non-mental quality. We might clarify our conceptions of psychiatric treatment rationale and method if we reviewed them in this light. If our psychotherapeutic attempts can be made in such terminology as happily will evoke the desired type of mental associative connections in the patient's own psyche, then the net result will fairly well coincide with the therapist's expectation. In proportion, however, as the patient's range of associational habits varies from that of the therapist, there may be an increasing ineffectiveness of his treatment method, due to this divergence.

One of the important factors in producing such a divergence is the varying facilitation or inhibition to which the patient's available associative elements are subjected by the fluctuations of his own affective-instinctive pressures. A specific example will explain this statement: A person in the throes of sea-sickness could not tolerate medical advice about the importance of over-eating; he might, however, accept with interest medical advice to conserve his energy by bed rest. Again if the patient's physiological state of balance produces homoerotic tensions, he will be the more likely to form delusions of homosexual implications in one's therapeutic efforts so that they should accordingly be adapted as far as possible to avoid this supplementation from the patient's own drives.

In short, it seems that it would be helpful, in the planning and the performing of our psychotherapy, for us to realize that the processes involved are not exactly as they are ordinarily imagined. The added factor to be evaluated does, it is true, make the situation somewhat more complicated, but it enables us to form a more rational conception about what is actually happening when we try to observe or to change the psychic phenomena of patients. It may also serve to remove some of that tinge of magical, quasi-superstitious connotation which has always pervaded the lay reaction to the mental life, and which is still implied in some of our own habitual, hence unthinking, attitudes about the psyche and its activities. But whether we propose to use organic therapy or psychotherapy,

we necessarily do actually administer to the patient something organic. We must content ourselves with the expectation that just as the body makes its own glucose when fed carbohydrates, so the brain will transmute this organic factor we administer into its own psychic elaboration. If we would use true psychotherapy, this is a way we can accomplish it.

New York Hospital—Westchester Division
White Plains, N. Y.

A REPORT ON A SUBCONVULSIVE REACTION TO ELECTRIC SHOCK AND ITS SEQUELAE IN A NORMAL SUBJECT

BY C. WATKINS, M. D., E. J. STAINBROOK, AND H. LÖWENBACH, M. D.

The behavior shown by human beings after therapeutic electric shocks suggests a state of temporary dissolution from which the personality then gradually reintegrates itself.[1] Our psychiatric and psychologic knowledge would be enriched by an adequate insight into the thoughts and feelings of a person during this restitution process. Unfortunately, we can never be certain how much the information obtained from psychiatric patients is contaminated by psychotic content, symbolism peculiar to the patient, and confabulation.

For various reasons, objections arise against experimental shock to normal subjects; and it was only after considerable deliberation that a volunteer was permitted to undergo this procedure. It was not an *a priori* impossibility that the uncertainty in the validation of introspective reports could be somewhat dispelled if the shock was given to normal individuals. However, no new pertinent subjective information was elicited from this voluntary subject, but some of the writers' objective observations seem worth reporting briefly, especially since experimental electric shock in nonpsychiatric subjects is rare.

C. W., a 25-year-old physician, submitted to a shock of 400 milliamperes, recorded by oscillograph, for .165 sec., which produced a minor reaction lasting a few seconds. This consisted of a partial raising of the body with a slight throwing-up of the arms, accompanied by some muttering and by definite and repeated spitting, and then a fumbling with the hands at the belt. Immediately afterward, the subject lay relaxed and was apneic for some seconds.

Rapport was reestablished after one minute and 30 seconds at which time the subject was asked to copy a *Gestalt* figure. His first reproductive attempt consisted of dextrad circling movements of small diameter for about 10 seconds, during which the eyes were staring and not fixed on the figure. This was stopped spontaneously; and he then tried to "close in" on the model and to draw almost directly on the figure, this time making an angular form

corresponding to the square part of the total *Gestalt*. This was interrupted by the experimenter, whereupon the subject proceeded to draw an adequate copy in the indicated space on the paper.

The Rorschach test had been given on the previous day; and in accordance with the writers' procedure with mental patients, the test was given again after the shock.

In general agreement with Kelley, Margulies, and Barrera, but in contradiction to the writers' findings in psychotic patients, no significant differences were observed in the experimental subject's Rorschach responses before and after the shock. Possible exceptions may be seen in a 16 per cent increase in the relative number of animal responses to the total number of responses and in the shift in the quantity of answers given to the last three color cards from 37 per cent of the total responses in the preshock record to 28 per cent in the postshock. The number of whole responses was decreased by three in the postshock record, and there was a similar decrease in responses to tiny details. Total responses in the first record were 59, as contrasted with 52 (exclusive of 90 repeated responses) in the postconvulsive results.

One mode of differentiation, the reaction to diffusion-shading of which there were two responses in the original record and which in a context of other signs may indicate a moody anxiety, disappeared entirely from the record taken after shock. Additionally, displayed in only two responses as an answer to the query as to the determinants of responses, there was a suggestion of a tendency to give more emphasis to color as a response-determinant. These last two differences, however, may have been quite accidental.

From the third to the ninth minute after shock, C. W. gave almost the same responses and these in the same general order as were given to the original presentation the day before the shock. The only substitutes in the first responses were "do-jigger" for the original "flying horse radiator symbol" and "bowling ball" for "bowling pin." Then at nine minutes the subject began to repeat responses which he had already given on the same card, and such repetition was particularly marked on cards IX and X. A total of 52 repeated responses was given on Card X which was finally taken from him. On account of this repetition, the succession of responses on cards V to X was somewhat irregular.

The subject's attention was not directed to his repetition and all during the test he behaved adequately in the total situation, cooperating willingly and spontaneously.

Forty-one minutes after the shock, when orientation as to time, place and person and memory was probed, it was found that C. W. had lost the ability of recalling recent as well as past events, and he could not retain information. He could name the place where he was, the town and the state, and the names of the persons with whom he was talking. He came very near to naming the year correctly, saying, "1940," but there his knowledge of contemporary time ended. He did not know the day nor the month nor the season, and it must be stated at once that he seemed quite unconcerned about it. His answers to the questions were, "I do not know;" and in no instance, at least during the first hour and a half, was this statement followed by a quest for information.

He was unable to retain knowledge of the correct date, forgetting it again a few minutes after it was given to him. That he was not confused may be shown by the following examples: (What month is it?) No answer. (Is it one of the warm months?) No answer. (Summer, fall or spring?) No answer. (Name all the months, please.) Does so in right order. (Right, now which is it?) "Well, it's one of the warm months." (It is February.) "Why would I have on a light suit?" Laughs annoyedly. "I wouldn't have on a light suit in February, would I?" (It is February.) "No." (It is February.) "It is?" This conversation occurred 53 minutes after the shock.

The subject inferred that it could not be winter from the fact that he was wearing a light suit; and this was to him a much more reliable inference than information given directly to him by another person. He reacted similarly when, three hours later, he inferred from a raincoat worn by one of the writers that it was raining outside, although he himself had just come in from a wet outdoors. When asked about the time of day, he said it must be evening because the lights were on, but he did not consult his wristwatch. In other words, he was able to make inferences but did not use adequate orientative methods.

That C. W. was unable to retain information was shown by the fact that one could repeat the date to him over and over again and it was immediately forgotten.

A fountain pen bought eight days previously was not recognized as his own when he accidentally discovered it in his pocket and was thought by the subject to have been put there by mistake. So, too, upon retiring four hours after the shock, C. W. went to a room which he had formerly occupied for many months instead of to the bedroom in which he had been sleeping for only a few nights. In this situation, too, he showed signs of disorientation only, and at no time was confused. The former room was immediately recognized as wrong because it was bare; and the new room was accepted as his because of the presence of personal belongings. A few minutes later, everything was doubted again, but could be reaffirmed by the same inferences.

About three hours after the shock when C. W. was asked how he felt, he described it as a "calm and cooling feeling;" and his behavior was that of a happy-go-lucky unconcerned person although no undue euphoria was exhibited. In addition, he showed no apprehension about his loss of memory and obviously had forgotten that he had forgotten.

The subject showed signs of anxiety for the first time 14 hours after the shock—upon awakening the following morning. He felt "queer" and had feelings of unreality, although he was at no time able to describe what he meant. He was not sure whether he had received a shock and reasoned that if he had had a shock, then everything was understandable and his state was simply the consequence of his having been shocked; but that if he had not received a shock, then his present state must be indicative of disease. He was easily reassured, and although he became progressively more aware of the gaps in his memory, he showed no other signs of anxiety and cannot recall any today.

During the following days, the lost memory gradually returned in the same manner as the recovery of memory after head traumas. At first, a few isolated happenings here and there were remembered and some situations recognized as having been experienced during this time. Their number gradually increased; and they could finally

be reconstructed into proper continuity. However, the events immediately before the shock and for 15 hours after the shock are completely blotted out.

From the experience in this one case, certain conclusions may be drawn.

Gillespie[2] reported the result of an inquiry into the subjective sensations of 23 mental patients who received metrazol injections. Because "the patients' account tended to be clouded by their mental condition," he himself also underwent a metrazol convulsion. Apart from the aura which does not exist in electrically-induced convulsions, he described distinctly unpleasant feelings with headache, anorexia and malaise for many hours afterwards. There also existed "some retrograde amnesia." The present subject, in contrast, had no somatic complaints whatsoever, neither spontaneously nor upon repeated questioning. Objectively, the amnesia with its resulting disorientation stood far in the foreground.

From the standpoint of the Rorschach technique, it is very significant that C. W. gave the same answers over and over again when he could not remember what responses he had given. He did not produce a single new response. This adds further strength to the assumption of the essential stability of the Rorschach method.

A long-lasting amnesia can follow a stimulation which produces only a minor reaction. In the 50 mental patients which the writers have treated and in cases reported in the literature, such long-lasting memory defects have been described only after several treatments some of which were generalized convulsions. The present experimental case demonstrates that the brain can react to an initial and only subconvulsive stimulation with such effects. Whether this is the type of reaction of the normal, as contrasted with the psychotic, or whether this is only an accidental observation of this one individual, the writers are not now in a position to state.

This same observation provokes speculation as to whether a long-lasting postshock disorientation of similar type constitutes, in a psychotic patient, a sign of a good prognosis. Certainly, the writers' experience has led them to believe that the probability of the patient's recovery or remission is small if within a very short time after the shock he displays all the characteristics of his pre-

shock behavior. Patients who are confused and disoriented after the shock usually clamor for help in orientation and are then, in the writers' experience, much more susceptible to psychotherapy.

A word seems in place here about the similarity of this normal person's postshock behavior to the epileptic twilight state. A casual observer would not have noticed that our subject was living intellectually from one minute to the next. (Actually the waiter and the cashier in a restaurant to which the subject was accompanied did not notice the state in which he was, although he was unable to visualize the items on the menu and therefore made "random" orders.) Here, as in the twilight state, it is remarkable that ". . . a man seems completely conscious and able to act with intention, even though often irrationally, and notwithstanding he is in a state of severe impairment of his mental function." (Aschaffenburg.)

Summary

An electric shock, with subconvulsive reaction, was given to a 25-year-old normal white man. His behavior and experience during the period of recovery, in which especially the Rorschach test was employed, are reported. Absence of somatic and mental complaints, and presence of amnesia and of disorientation are described as the outstanding features and are briefly discussed.

Department of Neuropsychiatry

Duke University School of Medicine, Durham, N. C.
Dix Hill State Hospital
Raleigh, N. C.

REFERENCES

1. Löwenbach, H., and Stainbrook, E. J.: Observations on mental patients after electroshocks. Proc. Amer. Psychiat. Assoc., Richmond, Va., May, 1941.
2. Gillespie, J. E. O. N.: Cardiazol convulsions, the subjective aspect. Lancet, 1939, I, 391-392.

ELECTRIC SHOCK THERAPY IN THE PSYCHOSES: CONVULSIVE AND SUBCONVULSIVE METHODS

BY SERGE ANDROP, M. D.

The use of electricity in the treatment of mental disorders is not new; we can trace it as far back as the middle of the eighteenth century when it was claimed that all mental diseases had been successfully treated by electricity. For over a century, electrical treatment was extremely popular and widely used, so much so that Huff[1] in 1853 insisted that "no nervous affection whatever should be regarded as incurable until electricity has in some form been tried." Since then, according to the brief résumé of Berkwitz,[2] Arndt in 1870, Allbutt in 1872, Erb in 1883, Hayness in 1884 and Blandford in 1886 all reported encouraging results from its use in the psychoses. Berkwitz further notes that "interest in electric shock treatment gradually waned and by the beginning of this century the treatment was no longer popular." However popular the use of electricity had been prior to that waning of interest, it had never been employed to the point of producing a major convulsion. Cerletti and Bini[3] introduced the treatment of the psychoses with electrically-produced convulsions in 1937. Their aim was to find a method which would eliminate all the dangers and unpleasant concomitants of pharmacological shock therapy and yet retain all its favorable results. Although electric shock therapy is only in its infancy, it has recently come into prominence, and has now gained wide acceptance and usage.

The present writer has used other methods of shock therapy with some degree of immediate success. However, in evaluating results,[4] it was stated: "It has long been known that shock may relieve mental illness, it is also known that severe illness may for a time favorably influence a case of schizophrenia or stop epileptic seizures. All of these effects, however, are transitory. In evaluating the results of shock therapy we are therefore concerned not so much with the immediate effects of the treatment as with its lasting effect, the permanency or duration of the improvement." The present report is based on a group of 50 patients who received over 1,000 electric shock treatments during a period of 12 months;

and although it is recognized that neither the number of patients or treatments, nor the elapsed time following the treatments, will allow a report of any degree of finality, it is felt that much valuable information has been gained, justifying this presentation. The group consisted of male patients, the great majority were chronic schizophrenics, with the duration of illness ranging from one to 15 years.

The Apparatus and Technic

There are various machines on the market for use in shock therapy; but, although they are manufactured by reliable firms it is suggested that the Council on Physical Therapeutics of the American Medical Association might well undertake a careful study of them, with a view to standardization in respect to safety, adequacy and accuracy. In view of the abundant literature and descriptive material available, it does not appear necessary to discuss in detail the apparatus used in this series of treatments. It is very simple and is a modification of the one used by Cerletti and Bini and described by Spiegel.[5] An alternating current is used which is the ordinary 50-60 cycle, 110 volt, city-supplied, house current. Without changing the type of current, it is put through an induction or variable voltage transformer and is brought to the desired voltage, while the time of exposure is controlled by a very accurate roentgen ray timer graded in one-twentieths of a second. The apparatus is equipped with adequate safety devices and is easy to operate.

The technic is equally simple; the resistance of the patient's head is determined routinely. The resistance or impedence, according to Krause[6] and his studies on dead mammalian heads, consists of the skin, muscles and periosteum, with the contents of the skull having no influence and the resistance being the same whether the skull is filled with brain or wet sponges. At first, the writer relied upon the resistance to determine the voltage to be used, basing this procedure on the theory that the greater the resistance, the greater the current necessary to overcome it. However, it was found in practice that as soon as electric current is applied, the resistance drops appreciably; this was also reported by Golla, Walter and Fleming,[7] Gonda,[8] Löwenbach, Androp and Lyman[16] and others. In the present series, the resistance dropped immediately upon the

application of the current from 40 to 80 per cent. Accordingly, determination of resistance became useless for the calculations. However, its determination is still continued, because it gives assurance of an adequate contact between electrodes and skin; unusually high resistance suggests a poor contact, extremely low resistance, a short circuit. The patient, dressed in loose garments, is placed on a lightly padded table, with a hard pillow under his dorsospinal region to help prevent vertebral injury. The electrodes are placed on the temples close to the hair line after an application of Cambridge electric jelly. A rubber band about the head holds the electrodes in place. The electrodes should be large enough to prevent local burns. The electrodes are placed on the temples, in order to affect the frontal and precentral regions of the brain and to avoid damage to vital centers situated posteriorly, as well as because the temples are devoid of hair. The instrument is then set for the desired voltage, and the current is applied. The determination of the convulsive threshold is purely empirical. It is done by an initial application in each new case of from 80 to 90 volts. If a convulsion results, this is designated as the convulsive dose for the patient; if the voltage is inadequate to produce a generalized convulsion and results in a petit mal attack, it is called the subconvulsive dose. According to various calculations only a small portion of the amperage actually reaches the brain. This is variously estimated at between 1 and 10 per cent. At subsequent treatments, the voltage may be increased or decreased depending on what results are desired.

The major convulsion needs no detailed description, as it resembles very closely an epileptic attack. The convulsion follows immediately the application of the current and, in the great majority of cases, there is an instantaneous loss of consciousness with no latent period. As a rule, the convulsion consists of only two stages, the tonic and clonic, the whole cycle lasting about 45 seconds; and it is the writer's impression that it is somewhat less in severity than that produced by metrazol. On the other hand, the stage of apnea and cyanosis immediately following the clonic stage may be unduly prolonged, causing some anxiety. However, in only a few cases, was it necessary to interfere by pressure upon the chest to stimulate respiration. This stage is followed by a short period of

stupor and sleep, then a period of confusion lasting for about 30 minutes, and then a complete amnesia for the treatment. Involuntary urination may occur if the bladder is not emptied before the treatment, no defecation or seminal loss was encountered. There is always a retrograde amnesia for a short period preceding the treatment, and there may be an anterograde amnesia for a very brief period following the treatment.

The petit mal attack which results from a subconvulsive electric dose resembles very much a light epileptic attack, minus the muscular convulsion. Any of the following symptoms in various combinations mark this reaction: instantaneous loss of consciousness, flushing of the face, dilatation of the pupils, twitching of groups of muscles or individual muscles, especially of the face. The eyes are wide open, and conjunctival and corneal reflexes are abolished or diminished. There are smacking and sucking movements of the lips and tongue, and the patient fumbles about his clothing with his fingers. The expression is that of bewilderment, and there is a complete retrograde amnesia which lasts in proportion to the severity of the reaction.

Complications, Hazards of Treatment, Contraindications and Possible Injury to the Central Nervous System

As already noted, very little is known of the actual amount of electricity that reaches the brain when a given amperage passes between electrodes placed over the temples, neither do we know of the exact amount of stimulation which goes to specific parts of the brain. The figures given by different observers vary, the consensus being that not over 10 per cent of the amperage applied to the temples actually reaches the brain. This may be considered one of the reasons for such a divergence of opinion as to the safety of this means of treatment and as to the possible injury to the nervous system. On the one hand is Cerletti, the originator of the method of treatment, who, reporting on 3,000 convulsions in more than 100 patients, says: "Fractures and dislocations never occur. Cerebral complications or mental sequelae have never been seen during or after the treatment with the two years application of the method. That there are no anatomical reasons to expect such complications is shown by means of extensive investigations on shock-

treated animals. Irreversible cell lesions are found in insulin-treated as well as in cardiazol (metrazol)-treated animals. They are never found in dogs treated with electric convulsion treatment; here only reversible changes such as Nissl's acute cell disease were seen." Bini,[9] a coworker of Cerletti, "asserted that the brain of a dog had tolerated, without any apparent damage, a current of 3,000 milliamperes. The animal died when the duration of the passage of the current was prolonged to 60 or 90 seconds. Bini believes that the duration of the passage of the current is more important from the point of view of any damage done to the tissue of the brain than the tension of the current. However, many physicians and physiologists who have had experience with such technics consider the assertion unbelievable, and feel that under any circumstances the passing of electric current through the brain is a most hazardous procedure." Kalinowski,[10] another coworker of Cerletti, asserts that "organic damage . . . in the brain . . . is obtained only with much higher voltages," i. e., than used in electric convulsive therapy.

Experts in electronics[10] maintain that "all evidence indicates that brain damage is possible with this form of shock therapy, just as it is with other forms, and must be considered in the giving of the treatments. The theory of electric shock therapy is but little understood. The centers which it is necessary to stimulate are unknown; the time intensity relationship of the electric current required for convulsion has not been thoroughly investigated, and little is known of the extent of brain damage."

Although there are claims that a wide margin of safety exists between the maximal dosage used in electric shock therapy and that which may produce damage, there are many who dispute that view. Berkwitz[2] in reviewing the technic and dosage of Cerletti and Bini, remarks that "a current of such high milliamperage and low frequency is apt to produce severe changes in the central nervous system." In connection with possible damage to the nervous system, an interesting problem arises, that of the time-intensity relationship to the damage. The question presenting is: Should we use a higher current for a short time or decrease the current and increase the time? Alexander[11] writes: "On the whole, the amperage factor is more important than the time factor; that is,

I should rather prefer lower amperage for slightly longer time than vice versa. Increasing the time by 200 per cent has about the same effect as increasing the amperage by 25 per cent. That is true for the peripheral nerve where I have studied it experimentally. On the whole I feel that amperage and time should be kept down to the indispensable minimum." Putnam[12] writes: "On general principles I should think that there was less danger with the lower voltages even though applied for a longer time, but I doubt if either method of application is dangerous. Certainly enormous amperages have been survived if the exposure has been brief." Gonda[8] holds that, "based upon theoretical considerations, it is advisable to increase the potential rather than the time." Other authorities[10] have considered it desirable to increase the treatment time as long as a considerable decrease in the required current results. In this connection, one must remember that the heating power goes up as the square of the current, so that doubling its strength is from this standpoint equivalent to quadrupling the duration. The present writer, after considerable experimentation, settled to a standard time of five-tenths of a second, varying the potential in order to get the desired results.

As to the probable damage to the central nervous system from electric shock therapy, there is a paucity of authentic reports in the literature. Even at this writing, however, considerable experimentation in this line on animals is going on. In this connection, the writer in collaboration with H. S. Rubinstein has undertaken a series of experiments at the Laboratory for Neuroendocrine Research, Surgical Division, Sinai Hospital. Apparently there is insufficient data yet available to determine whether irreversible changes are produced in patients following repeated electric shocks; and there is great need for careful histopathologic studies of the central nervous system, especially the brain, following repeated passage of electric current, under varying conditions. Although Cerletti,[8] in his report of 3,000 convulsions, states that fractures and dislocations never occurred, these complications are beginning to appear in the latest reports. Bingel and Meggendorfer[9] report dislocations of the jaw and of the shoulder and fractures of the humerus and scapula. Flemming[8] reports two cases of compression fracture of dorsal vertebrae among his first 20 cases. Gonda[8] en-

countered a fracture of both humeri in a woman. Smith, Hughes and Hastings[13] report compression fracture of a thoracic vertebra and dislocation of the shoulder in a single patient. Recently, several fractures of long bones came to the present writer's attention through verbal communications. In the present series, three mandibular dislocations and no fractures were encountered. It is hoped that fractures and dislocations may be minimized by holding up the lower jaw, and holding down the upper and lower extremities during the convulsion. The possibility of shoulder dislocation and humeral fracture seems greater in electric shock than in other forms of shock treatment because of the tendency of the patient toward arm extension over the head at the start of the seizure. It has been previously suggested that a hard pillow be placed under the dorsal spine to prevent vertebral compression fractures.

Although, so far, no fatalities have been reported as a result of electric shock treatment, we must ever bear in mind the possibility of such occurrences. Alexander[14] warns that the fatal voltage may be remarkably small, even as low as 25 volts, if the resistance is sufficiently lowered by perfect contact and long duration of flow of current. He says that the causes of death in immediate connection with an electric trauma are cardiac and respiratory failure. Cardiac failure may be disregarded to some degree in electric shock therapy; for it has been shown when the current is passed from temple to temple, the heart is either not included in the field, or the percentage of current passing through the heart is greatly reduced. Although the chances of respiratory failure may be remote, it is best to bear them in mind and be prepared for emergencies should they ever arise. Jellinek[14] emphasized that "death from electric shock is not immediate, but delayed and slow; that the state of apnea and of apparent standstill of circulation following electric shock is due to prolonged but not permanent paralysis of the respiratory vagus and pressor centers in the medulla oblongata; and that during this period the individual remains viable and resuscitable for a surprisingly long time, provided that artificial respiration is started as promptly as possible and is adequately carried out for a sufficiently long period of time." MacLachlan[14] describes a case in which the patient was successfully revived after eight hours of prone pressure resuscitation. Accord-

ingly, Alexander[14] advocates the following procedure in the treatment of acute shock from electricity: artificial respiration by the prone pressure method, started immediately and carried out untiringly if necessary for 12 hours; intracardial injections of atropine and adrenalin followed by lumbar puncture for relief of increased intracranial pressure, repeated if necessary on subsequent days.

As to contraindications for this method of shock therapy, it may be said that in general the same conditions which preclude the use of metrazol should be considered in the use of convulsive doses of electric shock. Acute infections and febrile diseases, active tuberculosis, advanced heart and kidney disease, atrophy of bones in patients who have been bedridden for a long time, pronounced curvatures of the spine and osteoarthritis, malignancy, severe thyrotoxicosis, thrombophlebitis and advanced generalized arteriosclerosis are all more or less contraindications for this treatment in convulsive doses. As it will be pointed out later, the majority of these contraindications may be overlooked in the treatment with subconvulsive doses. Needless to say, each patient should receive a thorough physical examination before the treatment is begun, including X-rays of the spine and an electrocardiogram if possible.

Subconvulsive Method

The exact *modus operandi* in the production of a convulsion by either metrazol or electric shock has not yet been definitely and satisfactorily explained. The earlier theories held that sudden vasoconstriction of the cerebral blood vessels explained the convulsion; the more recent views lean toward a direct cerebral stimulation as the mechanism that initiates the fit. Clark and Wall[15] held that direct cerebral stimulation and not vasoconstriction of the cerebral vessels was responsible for the convulsions, "since stimulation of the proximal end of the cervical sympathetics, while producing vasoconstriction did not elicit an epileptic seizure." Others have also showed that with the hemispheres removed, stimulation of the midbrain produces attacks.

Neither has the exact mechanism responsible for the beneficial results obtained with shock treatment in the psychoses been agreed on. There are numerous theories to explain these results. There

are those who maintain that it is sudden severe anemia and the immediately following hyperemia to the brain which benefit the patient. Others hold that the resultant apnea produces an accumulation of carbon dioxide which is immediately and vigorously neutralized by large amounts of air, and that this process is responsible for the changes which are observed. Still another group maintains that during the violent metabolic process unknown "toxic" substances are neutralized or destroyed. And more recently there are claims that the beneficial results are produced by profound changes of the cerebral electrical activity, as shown by the electroencephalogram. Regarding this, Gonda[8] states that heretofore "it has been a common interpretation that the electricity acted in a purely 'suggestive' manner in these cases. If, at that time, one could have had the temerity to suggest that the electricity acted as an agent to reverse reversible changes in the nervous system, or to create biochemical changes, he would have been called unscientific. In view of the latest developments regarding electrically-induced convulsions, we may soon have to revise our conceptions on this important question." The disappearance of the superficial and deep reflexes immediately following the convulsion, later the exaggeration of the deep reflexes and the appearance of pathological signs such as sustained and pronounced ankle clonus, less frequently patellar clonus, positive Babinski, Hoffman and grasp reflexes with persistent sucking—all of which soon disappear—testify to a temporary but profound involvement of the pyramidal and extrapyramidal motor systems. Is it the biochemical mechanism of the convulsion that is the important factor or the profound changes of the electroactivity of the brain? In a group of 10 patients to whom electric shock therapy was administered by Löwenbach, Androp and Lyman,[16] electroencephalograms of the patients before, during, and after the convulsion were recorded. "In the E. E. G., the major convulsion starts with an unbroken run of spikes of huge amplitude, somewhat obscured by muscular action currents. Gradually they make room for groups which resemble spikes and waves to a certain extent. The continuous row of spikes coincides with the tonic phase, the 'almost-spikes-and-waves' with the clonic phase. Toward the end of the clonic period the E. E. G. becomes flatter, and finally a silent period of several seconds dura-

tion leads over to the rebuilding of an organized pattern. The time needed for this usually does not exceed 30 minutes. No essential differences exist compared with the curves obtained from shocks induced with metrazol."

Löwenbach and Lyman[17] administered electric shock to a series of rabbits and observed the cerebral activity during and following the application of the current. It was observed that profound changes of the electroactivity of the brain, sometimes lasting through several minutes, took place when the current merely threw the animal into a state of unconsciousness and areflexia, with the avoidance of convulsive phenomena. Following this, Löwenbach, Androp and Lyman[16] treated 10 patients with a total of 140 shocks. "In all of them it was attempted, whenever possible, to avoid convulsions and to induce profound but transient changes of the cerebral activity, of which we made sure by simultaneous electroencephalography, and thus to spare the patients the violent muscular contractions of the fully-developed attack and to avoid the dangers of the severe convulsion. This subconvulsive attack was accompanied by marked changes of the pattern of the E. E. G. Large 3-5 second waves were present, usually in all regions, foremost in the frontal ones. Never did we, interestingly enough, encounter in these purely petit mal reactions the 'spike and wave' runs which Gibbs and Lennox[18] have demonstrated to have pathognomonic significance in the 'genuine' petit mal. With the gradual return of the patient into consciousness, the E. E. G. equally returns to its original pattern. Sometimes we have seen noteworthy changes over one region only." It was also observed that if the current is insufficient or the time too short, only a slight motor reaction follows, there is no amnesia, the patient is fully conscious of the whole procedure, against which he usually protests. When there is this reaction, the E. E. G. does not show any changes except for transient interference by mechanical movements of the patient and the absence of alpha waves for an often considerable time.[16] This was called the "psychological reaction," and there was an attempt to avoid it in the present series. By the introduction of subconvulsive shock therapy, it was sought to answer the vital question as to whether the muscular convulsions that accompany shock therapy are essential for its therapeutic success.[19]

The introduction and use of curare was another attempt to answer this question. Even as far back as 1936, Meduna[20] wrote: "It is necessary to try other substances as well as to search for the factor mobilized by the convulsion and causing its beneficial effect. In this way it will perhaps become possible to eliminate the convulsion and apply only its secondary effects."

Results

While the value of shock therapy in acute cases (less than six months duration) has been admitted, its value in chronic cases has been questioned. The 50 cases which are presented here are all moderately chronic—of from one to 15 years duration. The average was about four years. Only one was of less than one year duration. The patients were all males and chiefly represented the various types of schizophrenia, with only a small proportion of the affective disorders. A total of 1,004 shocks was administered. Three hundred and fifty-four resulted in convulsions, and 650 were of subconvulsive dosage, resulting in petit mal attacks. The elapsed time since the completion of treatment is from one to 12 months. Of the total patients treated, 48 per cent showed no perceptible improvement. A total of 52 per cent improved—44 per cent social improvements, 6 per cent institutional improvements, with only 2 per cent remissions. The criterion for the various types of improvement is based upon a table given in a previous publication.[4] Improvement of patients treated with convulsive doses reached the 60 per cent mark, while those treated with subconvulsive doses showed only a 46 per cent improvement. At the end of the first year, a total of 15 per cent had relapsed. The group treated with subconvulsive doses showed an 18 per cent relapse rate while those treated with convulsive doses showed only a 10 per cent rate. A similar group of patients who did not receive any form of shock therapy showed a 33 per cent rate of improvement during the first year of hospitalization. All the percentages given are based upon the small group of 50 patients and, therefore, are not put forward as absolutely accurate. However, these figures strongly indicate the general trend. Other suggestive impressions were as follows: A relationship between the rate of improvement and the duration of illness definitely exists. The convulsive doses

produce a higher percentage of improvement. Several subconvulsive treatments are required to obtain the therapeutic results of one convulsive treatment. The highest percentage of improvement was attained in those receiving intensive psychotherapy in conjunction with the shock therapy. The affective disorders gave the best results, and schizophrenics with affect responded better than those lacking affect. Those who had developed a more or less deep delusional system proved very resistive to improvement from shock therapy. Of course, the paranoid were the most resistive. The subconvulsive doses were given in elderly patients, and where convulsive treatment was contraindicated, with no untoward effects. In general, better results were obtained in those treated twice weekly than in those treated three times a week, and still better results were obtained in a group treated individually and symptomatically as the treatments were indicated and not according to a prearranged and unvaried schedule.

Discussion and Conclusions

Since the electric method is only one of several methods of shock therapy in the psychoses, it becomes necessary, for its proper evaluation, to answer three distinct questions:

1. How does it compare with other methods of shock therapy, especially with metrazol, as to administration, complications, contraindications and possible damage to the nervous system?
2. How does it compare in therapeutic results?
3. Does it constitute a cure?

There is no doubt that the electric shock method has many advantages: It is simple and economical. There is no dependency on veins, and such complications as thromboses of veins are avoided. The aura or latent period, with the accompanying horrifying feeling, is absent. Unconsciousness is instantaneous in a great majority of cases, and although latent periods of over a minute were encountered, these periods were covered by complete amnesia. Although there are reports of dislocations and fractures, the convulsion appears to be less violent and it may, therefore, be expected to have the rate of fractures much reduced over that of metrazol. In the series here presented, there were three mandibular dislocations

and no fractures. Both toxic and cumulative effects are eliminated, as well as the possibility of a succession of fits. The patients seem to tolerate electric shock better, and the writer met with much less objection to this method than to metrazol. The statement made by some writers that patients like the treatment is, in the present writer's opinion, a great exaggeration. Only one patient in this series actually asked for the treatment. Every patient was questioned upon completion of the treatment, and everyone expressed dislike for it which varied only in degree. Of course, it is necessary to distinguish between those who fear and dislike the procedure and those who refuse it because of their schizophrenic negativism and who would refuse any other method of treatment for the same reason. This method appears more suitable in relapsing cases and where prolonged treatment with maintenance doses becomes necessary. It is ideal in the treatment with subconvulsive doses. These same doses with metrazol were deleterious in that they left the patient fearful and apprehensive. A negligible number of fatalities have been reported with metrazol therapy, and none so far with electric shock.

Although it has been stated that[21] "it is always possible to produce an attack once the convulsive threshold has been determined," it was found that the patients developed a degree of tolerance to the treatment as the convulsive threshold moved up, and the dosage had to be progressively increased to overcome this and obtain a convulsion. This varied considerably and in a few cases it was necessary to increase the potential from 10 to 30 volts in the course of 20 convulsions. It was the observation of this which prompted Rubinstein to suggest, at least on theoretical grounds, that electric shock could be used in the treatment of epilepsy. Since the patient acquires a certain degree of immunity to the treatment as it progresses, and since the convulsive threshold is progressively raised, requiring higher currents to produce a convulsion, theoretically it becomes plausible to raise the epileptic threshold of the patient to the level of immunity or absolute resistance to the phenomena which precipitate an epileptic fit. This theory needs further investigation. Several workers report that if a convulsion is not obtained by the initial dose, a second shock is administered, with either an increased potential or increased time, immediately follow-

ing, and even a third until a convulsion is obtained. This procedure was never followed in this series on theoretical grounds; if a petit mal, with complete amnesia, was elicited, the patient was not treated again until at least the following day. Experience proved that the petit mal attack was beneficial to the patient, and at least it is harmless and has not the contraindications of the metrazol subconvulsive dose. It was previously stated that immediately following the application of the current, the resistance drops from 40 to 80 per cent. Alexander[14] warns that "since the amperage of the current is the quotient of the voltage divided by resistance, the fatal voltage may be remarkably small, even as low as 25 volts, if the resistance is sufficiently lowered by perfect contact and long duration of flow of current." To be on safe grounds, therefore, the writer never immediately repeated the procedure.

As to the number of shocks constituting a series of treatments, it should be said that it is entirely empirical and should be governed by the indication as presented in each individual case. In general, it is advisable to treat the patient fully until it is felt that the peak of improvement is reached, bearing in mind that overtreating must be avoided as much as undertreating. In case a patient has not received sufficient treatment to hold the gain made, maintenance doses may be given once weekly, every two or three weeks, or as and when the patient appears to relapse, until the improvement is well maintained. Close contact must be maintained with the patient to achieve this goal.

As to fear and anxiety playing a rôle in the treatment, as they are said to do in metrazol therapy, it can be stated that although these elements are reduced to a minimum in electric shock therapy, we cannot entirely disclaim some psychic or emotional genesis for the influence of the treatment. Several indications of this were noted in the present series, but only two cases will be cited briefly. W. W., a schizophrenic, has suffered from extremely unpleasant and annoying noises in the left ear for over five years. No organic basis for this symptom was discovered after repeated examinations. Psychotherapy failed entirely to bring any relief. The patient heard of electric shock treatment, and through his sister requested the therapy for the relief of this annoying symptom. Although it was felt that the treatment would not succeed, the

writer yielded. After six subconvulsive doses the patient announced that the noise had moved into the right ear. After 10 more treatments the noise disappeared entirely, and the patient has been happy and free from it for the past eight months. Berkwitz[2] may have the explanation when he says "practically none of the writers who have written on shock therapy considered the importance which suggestion plays in the treatment. With the reassurance that the shock treatment will benefit the patient in spite of its unpleasant nature, indirect suggestion is definitely active. The patient has concrete and visible evidence that something is being done for him. It is a foible of human nature which assures the acceptance of concrete things with greater readiness than mere assertions and persuasions of a physician." Another instance is that of G. W., another schizophrenic, who refuses to eat—sometimes for two or three weeks—necessitating tube-feeding. When shock treatment is given at the start of a hunger period, he abandons the hunger and begins to eat. Metrazol accomplished the same results in this case.[26] The fact is interesting that subconvulsive doses will achieve the same results as convulsive doses, and sometimes the mere mention that treatment will be resorted to if the man does not resume eating will accomplish as much.

Other workers[13] have called attention to a persistent symptom of memory defect in this form of therapy. In the present series, this was noted frequently and lasted from minutes to hours as a general rule. Duration depended on the severity of the reaction, the symptom was reversible in all cases. Another observation relates to a frequent reversal of affect which is also reversible after a short period of time. As in pharmacological shock, it is not always certain what dosage will produce the original convulsions; there exist, as yet unexplainable, individual differences in convulsive thresholds; it also appears that differences in room temperature, humidity and barometric pressure may have some influence upon the convulsive threshold.

As to the comparison of therapeutic results in electric and pharmacologic shock therapy, it may be said that in general the electric method has produced any and all of the benefits attained with chemical shock. As to statistical data we may compare a 50 per cent improvement reported by this writer and another[4] in a group of 82

patients with psychoses of over four years duration after metrazol shock therapy, with the present report of 52 per cent improvement after electric shock therapy in a similar group of patients with an average duration of psychoses of over four years. Two years after metrazol therapy, 50 per cent of the originally improved patients had relapsed. One year after electric shock therapy, 15 per cent of the improved patients had relapsed.

The final question as to whether electric shock therapy constitutes a cure in the treatment of the psychoses meets with an emphatic reply in the negative. Although there are some who claimed 80 per cent cures in insulin, 90 per cent cures in metrazol, and now suggest 100 per cent cures in electric shock therapy, the real cure—as an ultimate in therapy of the psychoses—has not been attained through any form of shock. However, shock therapy has become one of our most successful adjuncts to psychotherapy. In contradistinction to the foregoing rather wild claims for shock therapy, there are workers who condemn it and advocate its abandonment. C. C. Birnie[22] of England in a discussion before the Royal Society of Medicine took a skeptical view. He objected to the use of the term "therapy" for what, he held was only an interesting piece of research. He took the same stand when metrazol therapy was brought forward.

We must not be swayed by either one of the two extremes, and should not commit the mistake of shelving convulsive shock therapy for the simple reason that it does not constitute a cure. The results obtained in the group presented in this paper bespeak its continuance and its careful use in some forms of the psychoses, in conjunction with psychotherapy, in order to procure the best results. It is interesting to note that the population in state hospitals has its highest tendency for (spontaneous) remissions or improvements in the first year after admission; this then declines abruptly and after the second year assumes a low, more or less stationary curve.[23] It is in this group of average duration—four years—in whom we expect very few (spontaneous) improvements, that rather gratifying results were obtained in this series. However, it must be emphasized again that the best results were obtained only in those cases where psychotherapy was employed.

Indeed, it is becoming more convincing that the various forms of shock therapy alone do not constitute a "cure" in the treatment of the functional psychoses. Berkwitz[2] believes "that their administration probably interferes with the various habit patterns of diseased thought processes and permits the therapist to replace them with healthy ideas of psychotherapy." Friedman emphasizes the need for reeducation of the patient by means of occupational, *milieu* and psychological therapies in conjunction with insulin and metrazol treatment. Even Sakel advocates psychotherapy as a necessary adjunct to shock therapy. Berkwitz[24] warns that "shock treatment may relieve the patient of acute psychotic symptoms, but it does not materially alter his constitutional make-up. For this reason it is understandable why relapses may occur among patients who have had remissions through shock therapy as well as among those who have had spontaneous remissions." Often the symptoms are a form of protection.[22] "The inhibitions, stupor and depression in many types of schizophrenia were attempts by nature to repress certain unconscious urges with which consciousness was unable to deal. The convulsive treatment released these urges, with which consciousness was no better able to deal than before the treatment."

Gonda[8] admonishes that "one cannot emphasize strongly enough the necessity for following the convulsive treatment with psychotherapy. It is not overstating the case to say that intensive psychotherapy is almost as important as the elicitation of the convulsion itself. This fact may become a great handicap to our large state institutions where relatively little time can be spent with each individual patient. Group psychotherapy may be of great help in overcoming this handicap." And in conclusion, Gonda[8] warns that "until the electrically-induced convulsions prove to be absolutely harmless by the test of time, they should not be used for treating those mental conditions like mild psychoneuroses which may be cured by other methods." May not the subconvulsive method be an answer to this problem? Berkwitz[2] reported encouraging results by treating a small group of patients with very mild "Faradic shock." He attributed the success to the suggestion that something concrete is being done, as well as to the fear, pain and emotional reaction created. According to Janet,[25] this is plausible for,

"The psychotherapist who understands his patient well and who knows how to use psychological stimulation, succeeds with any method he cares to use." However, the patients studied here who did not receive large enough doses to produce amnesia failed to respond favorably to the treatment.

The search is on for a method that would eliminate the muscular convulsion with its dangers and yet produce the desired results by a change of cerebral activity, as indicated by the electroencephalogram. Meduna[20] himself was looking for such a method when he wrote "Far from considering the convulsion therapy as the only possible way of curing schizophrenia, I think that it is only a first step on the as yet unaccustomed biologic road of influencing the schizophrenic process. It is necessary to try other substances as well as to search for the factor mobilized by the convulsion and causing its beneficial effect. In this way it will perhaps become possible to eliminate the convulsion and apply its secondary effects." It is, therefore, here suggested that the technic of treatment with subconvulsive doses, as here described, be applied to large groups of patients. At the present state of our knowledge regarding electric shock therapy, it is impossible to predict which one of the three methods is indicated in any individual case. The subconvulsive method should be used in all cases in which convulsive doses are contraindicated, and in the present series has produced gratifying results and has proved by far more efficacious than "psychological shocks." The present group is comparatively small and the time elapsed too short to offer any definite conclusions. The results are so encouraging, however, that further extensive trial of this technic is recommended in the hope of finding an efficacious method of shock therapy which will eliminate the muscular convulsion with its dangers and unpleasant concomitants, yet retain all its favorable results.

Summary

1. A brief review of the literature on electric shock therapy is presented.

2. Fifty cases of moderately chronic psychoses received over 1,000 electric shock treatments in convulsive and subconvulsive doses; and the technic, complications, hazards of treatment, contraindications, results, and other observations are discussed.

3. A comparative study between electric shock and pharmacologic shock therapy as well as between two methods of electric shock therapy, i. e., the convulsive and subconvulsive, is presented.

ACKNOWLEDGMENT

The writer wishes to acknowledge the aid of Dr. H. S. Rubinstein in connection with this series, in the actual work of administering the treatments, as well as in the giving of valuable advice.

Spring Grove State Hospital
Catonsville, Md.

REFERENCES

1. Huff, G.: Electrophysiology. New York, D. Appleton and Company, 1853.
2. Berkwitz, Nathaniel J.: Faradic shock treatment of the "functional" psychoses. The Journal-Lancet, Minneapolis, 59, No. 8, August, 1939.
3. Cerletti, U., and Bini, L.: Un nuovo metodo di shockterapia "L'elettroshock." Boll. Accad. Med., 136-138, Roma, 1938.
4. Androp, Serge, and Green, Ethel: Résumé of metrazol-shock therapy in schizophrenia. Dis. Nerv. Sys., 2:7, July, 1941.
5. Spiegel, E. A.: Quantitative determination of the convulsive reactivity by electric stimulation of the brain with the skull intact. Jour. Lab. and Clin. Med., 22:1274-1276, 1936.
6. Krause, W.: Über den Wechselstromwiderstand des Säugetierschädels. Dissertation, Leipzig, 1936; quoted from Zentralblatt f. d. ges. Physiologie 102, 38.
7. Golla E., Walter, W. G., and Fleming, G. W.: Electrically induced convulsions. Proc. Roy. Soc. Med., 33:261-267, 1940.
8. Gonda, Victor E.: Treatment of mental disorders with electrically-induced convulsions Dis. Nerv. Sys., 2, 3, March, 1941.
9. Electrical convulsion treatment of mental disorders. Editorial, J. A. M. A., August 10, 1940.
10. Electric Shock Therapy. Offner Electronics, Chicago, November, 1940.
11. Alexander, Leo, Harvard University Medical School. Personal communication, September 14, 1940.
12. Putnam, Tracy Jackson, Neurological Institute, New York. Personal communication, September 16, 1940.
13. Smith, Lauren H., Hughes, Joseph, and Hastings, Donald W.: First impressions of electroshock treatment. Penn. Med. J., January, 1941.
14. Alexander, Leo: Electrical injuries to the central nervous system. Medical Clinics of North America, Boston number, May, 1938.
15. Clark, G. A., and Wall, T. F.: Unconsciousness produced by electric currents. Quart. J. Exper. Physiol., 24:85, February, 1934.
16. Löwenbach, Hans, Androp, Serge, and Lyman, Richard S.: Subconvulsive and convulsive electric shock controlled by the electroencephalogram in psychotic patients. To be published.
17. Löwenbach, H., and Lyman, R. S.: The electroencephalogram in electrically-induced convulsions of rabbits. In press.
18. Gibbs, F. A., Lennox, W. G., and Gibbs, E. L.: The E. E. G. in diagnosis and in localization of epileptic seizures. Arch. Neur. and Psychiat., 36:1225-1235, 1936.
19. von Meduna, L.: The significance of the convulsive reaction during the insulin and the cardiazol therapy of schizophrenia. J. Nerv. and Ment. Dis., 87:133-139, 1938.

20. de Meduna, L.: New methods of medical treatment of schizophrenia. Arch. Neurol. and Psychiat., 35, 361, 1936.

21. Almansi, Renato, and Impastato, David J.: Electrically-induced convulsions in the treatment of mental diseases. N. Y. S. J. Med., 40:17, September 1, 1940.

22. Electrically-induced convulsions. Foreign Letter. J. A. M. A., 114, 10:903, March 9, 1940.

23. Preston, George H., commissioner of Maryland Department of Mental Hygiene, Personal communication.

24. Berkwitz, Nathaniel J.: Treatment of mental disorders with pharmacological and electrical shock methods. Minnesota Med., 24:25, January, 1941.

25. Janet, P.: Principles of Psychotherapy (translated by H. M. and E. R. Guthrie). Macmillan, New York, 1924.

26. Androp, Serge: The effect of metrazol administration on prolonged refusal of food in the psychoses. Am. J. Psychiat., 96:6, May, 1940.

ARTIFICIAL FEVER THERAPY IN GENERAL PARESIS WITH ELECTROENCEPHALOGRAPHIC STUDIES*

BY A. E. BENNETT, M. D., PAUL T. CASH, M. D., AND CLARENCE S. HOEKSTRA, M. D.

The first near-specific scientific therapeutic measure in psychiatry was Wagner Jauregg's treatment of general paresis by malarial inoculation. For a number of years, this method was accepted as truly specific. Then, however, other workers began to challenge its specificity and to report similar results by other fever-producing agents. Certain investigators, led by Bessemans,[1] followed by Schamberg and Rule,[2] Simpson,[3] Boak, Carpenter and Warren,[4] and others advanced experimental evidence that the treponema pallidum was destroyed in local lesions and markedly inhibited in systemic lesions by temperatures of 105.5° to 106° F. Clinical and experimental evidence has gradually accumulated to show that increased body heat, however produced, is the effective therapeutic agent which arrests active neurosyphilis.

Certain pioneers became interested in physical agents as safe, efficient methods of influencing the course of syphilitic infection. Mehrtens and Pouppirt,[5] in 1929, reported successes with hot baths. Neymann and Osborne,[6] in 1929, first reported a method of generalized fever induction by diathermy and reported 68 per cent of clinical remissions with an additional 8 per cent of improvements in 2[illegible] cases of general paresis. Since that time, many other investigators of a variety of fever-inducing agents, biologic, infectious, and physical have also reported results equal or superior to malarial fever treatment.

This cumulative evidence indicates that malarial therapy is not specific and that the common denominator of all the methods under discussion is increased body heat adversely affecting a thermolabile organism.

Neymann[7] summarized the results of 967 cases which were treated by physical artificial fever and reported in the literature up to about 1938. Included were the results of 26 clinics. Of those cases, 263, or 27 per cent, obtained complete clinical remissions,

*Read before the Illinois Psychiatric Society, Chicago, April 3, 1941.

while 351, or 36 per cent, were improved; deaths were 2 per cent. These experiences were reported at a time when fever therapy was still experimental and many refinements of technic were lacking. The results obtainable today are superior.

O'Leary,[8] an advocate of malarial therapy, asserted, in 1937, that in syphilitic meningo-encephalitis, artificial fever produced less favorable results than malaria, a 27 per cent remission rate as compared to 35 per cent remissions with malarial inoculation. Yet, in 1940, O'Leary headed the report[9] of the cooperative clinical groups in a comparative study of malarial and artificial fever, in which he apparently reversed his opinion. The conclusions of this group were that both forms of treatment show over 50 per cent improvement in mild paresis, and about 20 per cent improvement in intermediate paresis. However, in severe paresis, malaria is ineffectual, as only about 1 per cent improve, compared to 10 per cent of remissions with artificial fever therapy. The admitted death rate from malaria was 13 per cent, as compared to 8 per cent from artificial fever. Furthermore, with artificial fever therapy, there were 70 per cent clinical remissions in mild cases and 37.5 per cent remissions in intermediate cases as compared to 51 per cent in mild cases and 24 per cent in intermediate cases with malarial therapy. These results would indicate, in both mild and severe general paresis, considerably greater chance for clinical remissions from artificial fever than from malarial therapy.

In Wagner Jauregg's last contribution,[10] received in a personal communication just prior to his death, he discussed the relative merits of malarial and fever therapy. Jauregg still did not believe the spirochaeta pallida to be thermolabile at temperatures the body could withstand. He admitted, however, the superiority of fever therapy. "Results show that after artificial fever treatment of paralytics there were remissions similar to those with malaria. Also many others have found that treatment of dementia paralytica with physically-induced fever gave a number of full remissions as great or greater than malaria. The number of improvements were equally as good." As additional advantages of physical heat, he found that height of temperature, length of fever, and rest periods could be regulated; hospitalization could be avoided; patients could continue their full occupations; and the effect of treatment was in-

nocuous; whereas malarial treatment required several weeks interference with work; and its danger was shown by cases of death. His conclusion was significant: "The best treatment of dementia paralytica is prophylactic energetic treatment of the latently positive spinal fluid cases found three to 15 years after infection. If unresponsive to chemotherapy then the patient should be treated by malaria or the artificial fever-chemotherapy method, the comparative value of which is still a matter of discussion."

After 10 years of experience with malarial therapy, the writers became convinced that the treatment was empiric and that some more scientific form of fever therapy would replace it. Since 1934, when the department of fever therapy research at the University of Nebraska was established, the writers have continued to treat all phases of resistant syphilis by means of combined artificial fever and chemotherapy. The means of fever induction has been the air conditioned cabinet or hypertherm. At the present time, this method, in preference to malaria, is used to treat all cases of general paresis in local, private, and state institutions in Nebraska.

The present report gives the results obtained by the method in the University of Nebraska fever therapy department and at the Hastings State Hospital. Preliminary electroencephalographic observations made in both untreated and treated cases are also reported.

TABLE 1. UNIVERSITY FEVER DEPARTMENT—CLINICAL RESULTS FROM COMBINED FEVER AND CHEMOTHERAPY IN 50 CASES OF GENERAL PARESIS

Severity:

41, or 82 per cent, were graded as mild or intermediate types
9, or 18 per cent, were graded as severe types
7 patients were taboparetics
1 was a juvenile paretic
40 were male, and 10 were female patients.

Degree of improvement:

	A (Full remission)	B (Improved)	C (Unimproved)	Deaths
Immediate:	20 or 40 per cent	26 or 52 per cent	4 or 8 per cent	1 or 2 per cent
Followups: (3 yrs.)	28 or 56 per cent	17 or 34 per cent	3 or 6 per cent	2 or 4 per cent

In the series shown in Tables 1 and 2, mainly private patients, only about half of whom were hospitalized for part of their treatments, 82 per cent were graded as mild or intermediate types of

general paresis. After an adequate course of artificial fever combined with chemotherapy, 56 per cent, on a three-year followup, were in full remission or had returned to former economic and social status, while an additional 34 per cent were improved.

TABLE 2. UNIVERSITY FEVER DEPARTMENT—SEROLOGIC RESULTS FOLLOWING COMBINED FEVER AND CHEMOTHERAPY

	Blood	Spinal fluid
Immediate:		
	Less positive	Less positive
	11 of 46—24 per cent	28 of 41—68 per cent
	Reversed negative	Reversed negative
	8 of 46—17 per cent	7 of 41—15 per cent
After 1 to 5 years:		
	Less positive	Less positive
	14 of 29—49 per cent	26 of 30—76 per cent
	Reversed negative	Reversed negative
	10 of 29—34 per cent	19 of 30—63 per cent

The followup serologic results of this group showed 34 per cent with negative blood studies, 63 per cent with complete reversal of spinal fluid, and 76 per cent with less positive spinal fluid tests.

TABLE 3. HASTINGS STATE HOSPITAL*—CLINICAL RESULTS FROM COMBINED FEVER AND CHEMOTHERAPY IN 79 CASES OF GENERAL PARESIS

Severity: (All were committed patients and many were hospital residents two or more years.)

37, or 47.3 per cent, were graded, as mild or intermediate types
42, or 52.2 per cent, were graded as severe types
11 patients were taboparetics
3 patients were juvenile paretics
57 were males and 22 were females; 11 were negroes, who were graded intermediate or severe types.

Degree of improvement:

A	B	C	
(Full remission)	(Improved)	(Unimproved)	Deaths
15 or 19 per cent	26 or 32.9 per cent	31 or 40.7 per cent	7 or 7.9 per cent

In 1937, when treatment was begun in the state hospital, the patients at first included a large number of chronic hospital residents, including malaria treatment failures. All patients in the institution with general paresis, many of these grave risks, were treated with artificial fever. (Table 3.) Of this group, in which

*The writers are indebted to Dr. Juul C. Nielson and his staff at Hastings State Hospital for their cooperation in furnishing records.

52.2 per cent were graded as severe types of general paresis and in which three patients were juvenile paretics, only 19 per cent of 79 cases followed as long as three years (Table 3) had full, complete clinical remissions, while 32.9 per cent more showed improvement, 40.7 per cent were unimproved, and 7.9 per cent died within three months of treatment. The final analysis of this group compares unfavorably with the university fever department series, in which 82 per cent of the cases were of mild or intermediate type.

The serologic reversals in the state hospital group were, likewise, less striking than in the series of private patients. After a three-year followup 15 per cent showed complete blood reversals and 42 per cent had spinal fluid reversals with 63 per cent of spinal fluids less positive. (Table 4.)

TABLE 4. HASTINGS STATE HOSPITAL—SEROLOGIC RESULTS FOLLOWING COMBINED FEVER AND CHEMOTHERAPY

	Blood	Spinal fluid
Immediate:		
	Less positive 14 of 76—18 per cent	Less positive 36 of 75—48 per cent
	Reversed negative 12 of 76—16 per cent	Reversed negative 18 of 75—24 per cent
After 1 to 3 years:		
	Less positive 21 of 52—16 per cent	Less positive 33 of 52—63 per cent
	Reversed negative 8 of 52—15 per cent	Reversed negative 22 of 52—42 per cent

From the beginning of the writers' investigation, they have been interested in treating patients who had previously received malaria without obtaining full remissions. Of the patients reported on here, 24 had had previous malarial therapy. In this group, 13 patients had been somewhat improved by malaria and were graded as mild or intermediate types, while 11 were graded as severe. From this group, after combined artificial fever and chemotherapy, 29 per cent showed full remissions and 29 per cent were improved, while 42 per cent were unimproved or showed progressive manifestations of the disease. From the writers' experience, it is felt that any patient, particularly one with the mild or intermediate type of general paresis, who fails to obtain a full remission from

malaria should have an adequate course of artificial fever therapy plus chemotherapy. (Table 5.)

TABLE 5. UNIVERSITY FEVER DEPARTMENT AND STATE HOSPITAL—CLINICAL RESULTS FROM COMBINED FEVER AND CHEMOTHERAPY AFTER MALARIA HAD FAILED TO PRODUCE FULL REMISSIONS IN 24 CASES OF GENERAL PARESIS

Severity:

13 were graded mild or intermediate types
11 were graded severe types

Results:

	A (Full remission)	B (Improved)	C (Unimproved)	D (Progressed)
Immediate:	6 or 25 per cent	8 or 33 1/3 per cent	9 or 37½ per cent	1 or 4 1/6 per cent
Followup:	7 or 29 1/6 per cent	7 or 29 1/6 per cent	3 or 12½ per cent	7 or 29 1/6 per cent

Along with Jauregg, the writers have long advocated treatment of general paresis and prevention of development by controlling the asymptomatic types of neurosyphilis. O'Leary has recommended malaria for any patient with a persistently positive spinal fluid not responding to chemotherapy. In 1937, he stated,[8] "Malarial therapy will reverse spinal fluids in 25 per cent of cases in which chemical agents have failed. A sufficient time has not elapsed to make a similar appraisal of the value of electric machines. However the early impression is that fever machines will not do as well."

Fever therapy was used in 15 cases of resistant asymptomatic neurosyphilis with so-called Wassermann-fast blood tests and paretic formulae in the spinal fluid. (Table 6.) Practically all had failed to improve during many years of chemotherapy. After 30 to 50 hours of fever therapy combined with various chemicals, 46 per cent had negative bloods, an additional 20 per cent were less positive, the spinal fluids were reversed to negative in 73.3 per cent, and 20 per cent more became less positive. In only one patient, was the spinal fluid serology unimproved. In the writers' experience, artificial fever therapy has been far superior to malaria in this group and absolutely without the hazards and the economic loss caused by malaria. None of these patients was taken from active occupation while under treatment.

TABLE 6. UNIVERSITY FEVER DEPARTMENT—ASYMTOMATIC NEUROSYPHILIS—SEROLOGIC RESULTS IN 15 CASES AFTER 30 TO 50 HOURS OF ARTIFICIAL FEVER AND CHEMOTHERAPY

Blood		Spinal fluid	
Reversed	7 or 46.6 per cent	Reversed	11 or 73.3 per cent
Less positive	3 or 20 per cent	Less positive	3 or 20 per cent
Unimproved	5 or 33.3 per cent	Unimproved	1 or 6.6 per cent

All except one patient had had previous chemotherapy. All except two had had intensive previous chemotherapy. The average duration of infection was 12 years; the shortest period two years; the longest 30. The chemotherapy used in conjunction with the fever treatment was mapharsen and bismuth. The serologic reversals were all immediate except in two cases in which further chemotherapy then produced reversal.

ELECTROENCEPHALOGRAPHIC STUDIES

Within the past year, the electroencephalogram has been used as a routine procedure in all cases of general paresis treated in the fever therapy research department of the university.

Disturbance of electrocortical function in syphilitic meningoencephalitis was first described by Berger in 1931.[11] In this preliminary report, he described normal findings in 10 treated patients, all of whom were markedly demented. One acute case showed the alpha activity to be variable in frequency and amplitude.

In a second report, in 1933,[12] he described the findings in 29 cases. In all these cases, the records were of low, variable voltage with many short alpha or long beta waves of varying frequency. Once the disease process was arrested, the electroencephalogram returned to normal, even when marked mental defect remained. This, he thought, indicated that certain cerebral apparatus were injured by the disease but that the general electrocortical functions returned to normal.

In 1935,[13] Berger described other changes, such as variation in amplitude and shape in some, with asymmetrical fluctuations in amplitude in others. In other cases, there was a marked slowing of the alpha waves, with great variability in their duration. In certain cases with focal lesions, inequality in amplitude between the two hemispheres was found.

In 1936,[14] Berger reported on untreated cases of general paresis which showed a marked variation in frequency. He considered that the alpha waves of short duration were evidence of irritation. This shortening of the alpha waves was called "Reiz-symptom" and its similarity to the excitation phase of avertin narcosis was noted.

Hoagland[15] studied general paretics during hyperpyrexia treatments. Of six severe cases, one a "very dilapidated" paretic, showed irregular alpha potentials. Four of the other advanced patients showed critical thermal increments of frequency increase, with temperatures higher than normal.

Finley[16] reported that the more severe the mental symptoms, the more likely the patient was to show slow waves. Patients with mild symptoms tended to show abnormally fast activity. He found that treatment might restore high-voltage, fast activity in those who had ceased to show it.

The present writers have studied 10 consecutive patients who have received adequate treatment, i. e., 50 hours of combined artificial fever and chemotherapy, two of whom previously had had malarial therapy. Although the acute process has been influenced, these patients have remained psychotic and require continued institutionalization. Out of this series, there was only one normal record and one borderline normal. The eight abnormal records were characterized, with only one exception, by extremely low voltage. Other outstanding changes were: the disturbance of the alpha activity in seven of the eight abnormal records; the increased incidence of low-voltage, fast activity; and the presence of slow potentials of low amplitude and infrequent occurrence. In six of the eight abnormal records, the greatest involvement was in the frontomotor and transfrontal leads. Of the 10 patients, seven were grade C and the remaining three were classified as grade B. All 10 were classed as C results. The positive spinal fluid serology in seven of the 10 remained unchanged; among these was the patient with a normal electroencephalogram. The other three had negative spinal fluid before artificial fever therapy was instituted.

The writers have also studied four cases of acute general paresis before treatment was instituted and one case in which a previous course of combined artificial fever and chemotherapy resulted

in only slight improvement. This last had electroencephalographic studies preceding and following the second course of treatment. Of the four acute patients, two were studied before and after treatment.

The characteristics of these five records were, first of all, the marked disturbance of the alpha activity. In all five, there were only occasional brief outbursts of alpha activity—which varied in amplitude and form and which appeared from the two hemispheres in an independent fashion. Other characteristics were the frequent occurrence of slow potentials of varying frequency; the increased incidence of low-voltage activity above 16 cycles per second; and the more marked disturbance in all frontal leads. None of the five records showed the extremely low voltage which characterized most of the treatment failures.

The three cases studied immediately following treatment all showed a marked increase in alpha activity of more constant amplitude, frequency and form and of more synchronous occurrence from the two hemispheres. A decrease of low-voltage activity above 16 cycles per second was noted and no slow potentials were seen in any of the three records. Every case showed an increase in amplitude. In all three cases, there was definite clinical improvement by the time treatment was completed and at the time these records were taken.

The finding of a definitely abnormal electroencephalogram in eight out of 10 treatment failures does not agree with the findings previously published by Berger. With the exception of one case, all of the writers' patients were studied from at least one to three years following completion of combined artificial fever and chemotherapy. The rather consistent findings, in eight of the 10 cases, of an extremely low-voltage record, a very low incidence of alpha activity of varying amplitude, frequency and form—or even complete absence of alpha activity—and the increased low-voltage, fast activity, with only infrequent slow potentials, would seem to indicate a common pattern, which develops over a period of time in treatment failures and is the electrical expression of an irreversible organic change. That this type of record does not always appear is undoubted.

In the acute cases, certain changes are seen which distinguish these records from the normal and from those of the treatment failures. The records are of moderate amplitude, with a marked disturbance of alpha activity, i. e., irregular amplitude and frequency in short outbursts with loss of synchronism of the two hemispheres, increased incidence of low-voltage, fast activity, and frequent slow, pathological potentials.

In contrast to the acute cases, the records from the treatment-failure group show extremely low voltage, with little or no alpha activity and only very infrequent low-amplitude, slow potentials.

The three acute cases followed through treatment displayed a return of alpha activity of much more normal appearance and increased amplitude, with less high frequency activity, and with a disappearance of the slow potentials. Although the records are not rigidly normal they approximate normal. These records may indicate that the presence and frequency of occurrence of slow potentials is a gross measure of the severity of the infectious process. Early in these cases, at least in a certain percentage, the changes are reversible and apparently indicate an acute process without organic destruction of tissue.

In both the acute paretic patients and the chronic failures, there is a marked disturbance of the alpha activity, with tendency to complete or nearly complete disappearance of alpha waves in the more chronic cases. The amplitude would also appear to give some indication of the duration of the luetic process. The acute cases have moderate depression of amplitude; and seven out of 10 of the treatment failures of longer duration have records of extremely low voltage.

The records of seven patients, out of the total 15 studied, showed the slow potentials to be of greater frequency of occurrence in the frontal leads.

Figure 1, left: This is a normal record, showing high per cent time alpha activity of good amplitude, form and of synchronous occurrence from the two hemispheres. There is little or no activity in the frontal and motor leads.

Right (H. S.): This is a record taken from a patient with acute general paresis. It is of a white male of 40 with spinal fluid strongly positive. In the F-O leads, there is alpha activity of vary-

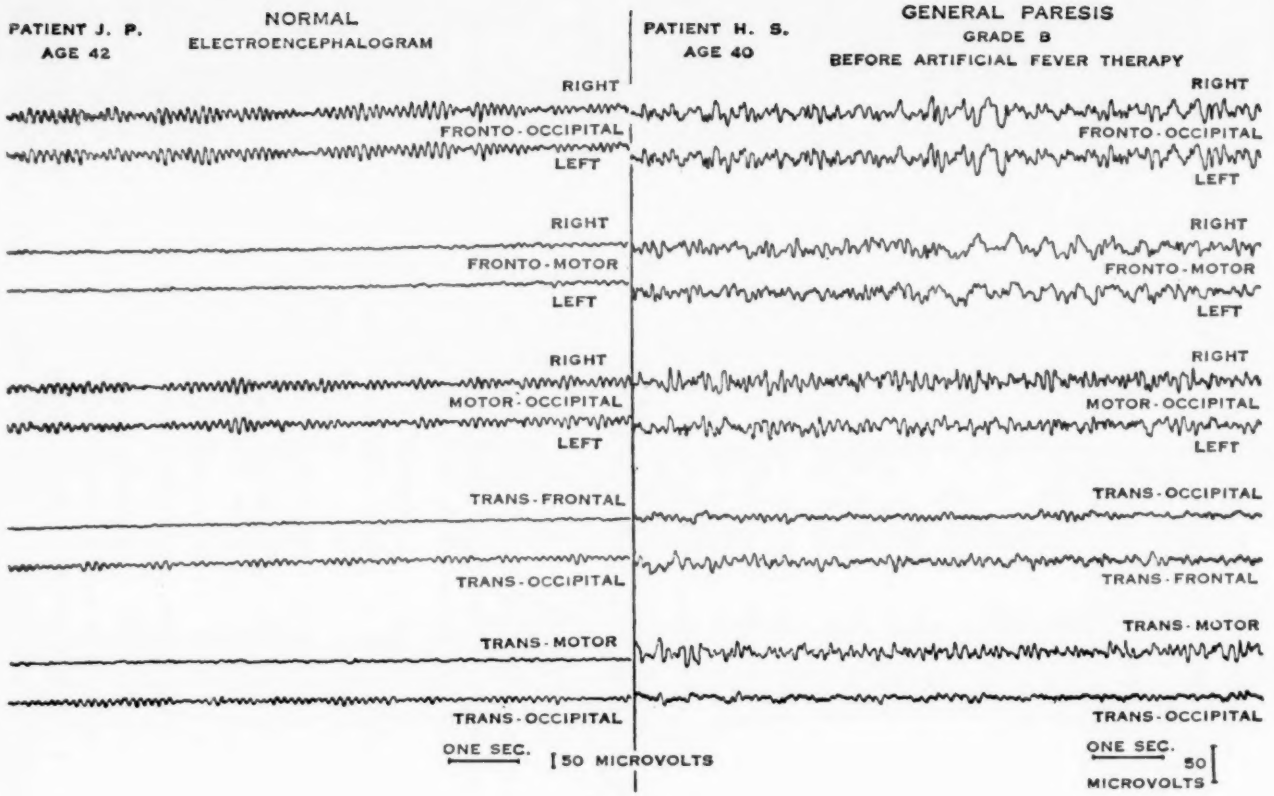

PATIENT J. P.
AGE 42
NORMAL
ELECTROENCEPHALOGRAM
PATIENT H. S.
AGE 40
GENERAL PARESIS
GRADE B
BEFORE ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS-FRONTAL
TRANS-OCCIPITAL
TRANS-MOTOR
TRANS-OCCIPITAL
ONE SEC.
50 MICROVOLTS
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS-OCCIPITAL
TRANS-FRONTAL
TRANS-MOTOR
TRANS-OCCIPITAL
ONE SEC.
50
MICROVOLTS

Figure 1

ing amplitude and frequency with frequent slow potentials. In the F-M leads, there is rhythmic activity of 8 to 9 cycles per second on the right, but to a much less extent on the left. There are also frequent slow potentials of about 3 cycles per second. In the M-O leads, there are only occasional low-amplitude, slow potentials. The alpha activity is of varying form, frequency and amplitude. The transverse leads show the greatest involvement to be in the frontal and motor leads.

Figure 2, left: This is a normal record.

Right (J. H.): The record is from a 40-year-old white male, a very early preparetic case. The spinal fluid report was necessary to establish the diagnosis. Blood and spinal fluid were strongly positive.

In the F-O leads, there is moderate depression of amplitude with a low incidence of alpha activity of varying amplitude, frequency and form. There are rather frequent slow potentials of 4 to 5 cycles per second of greater incidence on the right. There is an increased amount of low-voltage, fast activity of 30 cycles per second on the left. The low-voltage, fast activity is more evident in the F-M leads and still of greater incidence on the left. The M-O leads show the disturbance of alpha activity (varying frequency, amplitude and form), with low-voltage, fast activity only on the left. The transfrontal leads show only low-voltage activity of irregular frequency and form. The transmotor leads show low-voltage fast activity. The transoccipital leads show the alpha activity to be of somewhat irregular amplitude and form.

Figure 3, left (E. M.): A colored male of 31 with general paresis, grade C, received a total of 50 hours of artificial fever therapy with .66 gms. mapharsen and 1.43 gms. bismuth. The blood and spinal fluid remained positive. The result was classed as a C. An electroencephalogram taken one and one-half years later shows a low-voltage record with marked asymmetry between the two hemispheres. Alpha activity of low amplitude is present on the right in the F-O and M-O leads and is of 10 cycles per second, with normal form and regularity; but on the left, there is a nearly complete absence of activity.

Right (M. L.): This is a colored female of 44 years with general paresis, grade B. She received a total of 49 hours of artificial

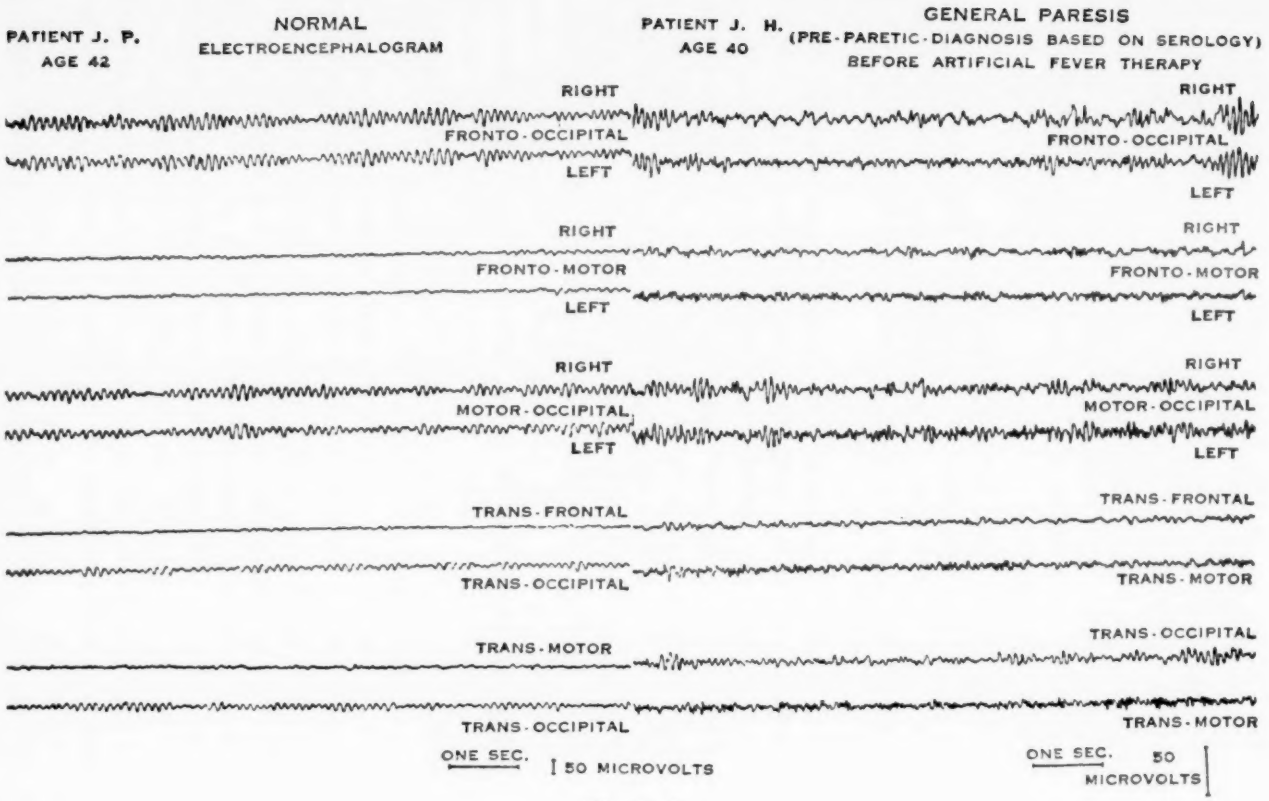

PATIENT J. P.
AGE 42
NORMAL
ELECTROENCEPHALOGRAM
PATIENT J. H.
AGE 40
GENERAL PARESIS
(PRE-PARETIC-DIAGNOSIS BASED ON SEROLOGY)
BEFORE ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS-FRONTAL
TRANS-OCCIPITAL
TRANS-MOTOR
TRANS-OCCIPITAL
ONE SEC.
50 MICROVOLTS

Figure 2

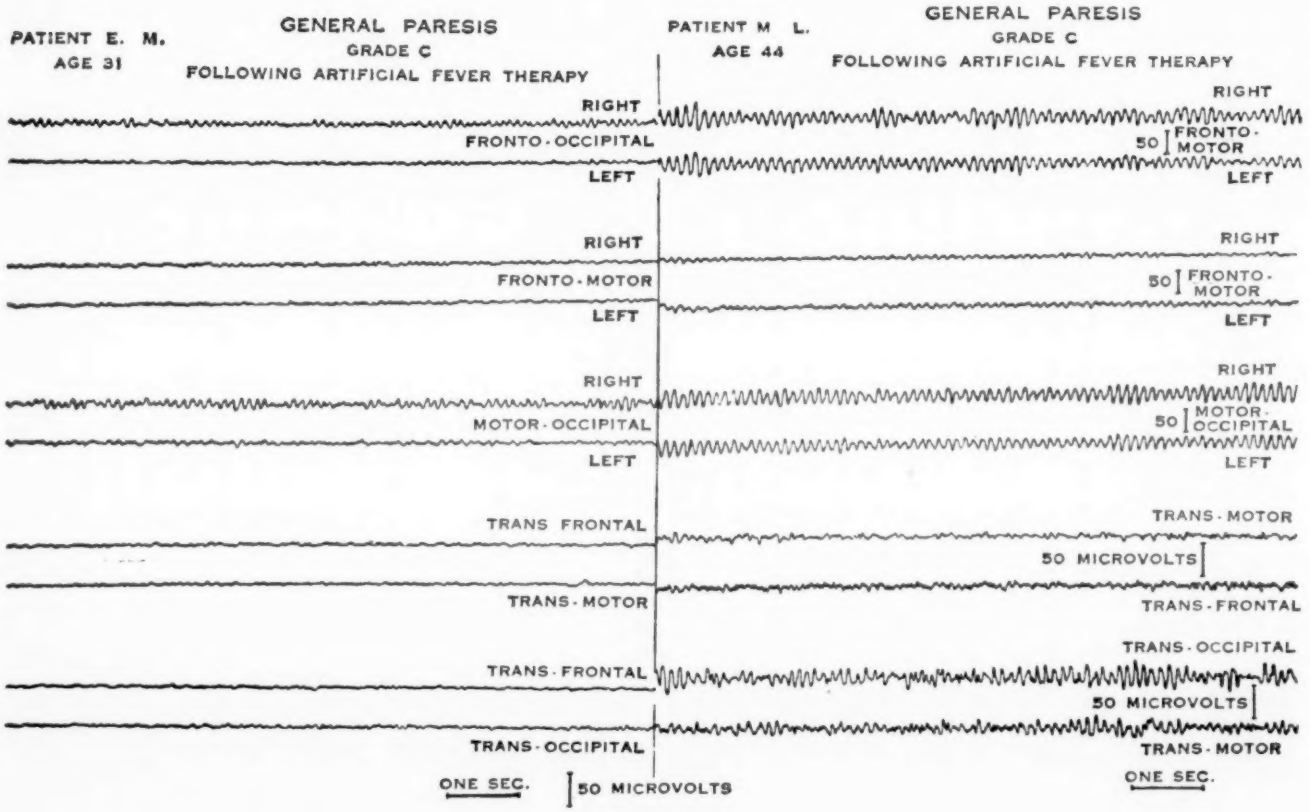

PATIENT E. M.
AGE 31
GENERAL PARESIS
GRADE C
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS FRONTAL
TRANS-MOTOR
TRANS-FRONTAL
TRANS-OCCIPITAL
ONE SEC.
50 MICROVOLTS
PATIENT M L.
AGE 44
GENERAL PARESIS
GRADE C
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
50 FRONTO-MOTOR
LEFT
RIGHT
50 FRONTO-MOTOR
LEFT
RIGHT
50 MOTOR-OCCIPITAL
LEFT
TRANS-MOTOR
50 MICROVOLTS
TRANS-FRONTAL
TRANS-OCCIPITAL
50 MICROVOLTS
TRANS-MOTOR
ONE SEC.

Figure 3

fever therapy with .96 gms. of mapharsen and 2.08 gms. bismuth. The blood and spinal fluid remained positive. The result was classed as a C. After two years the electroencephalogram is normal.

Figure 4, left (J. L.): A colored male of 54 years with general paresis, grade C, received a total of 50 hours of artificial fever therapy with .90 gms. mapharsen and 1.95 gms. bismuth. The blood and spinal fluid remained positive. The result was classed as a C. The electroencephalogram, taken one and one-half years later, shows an extremely low-voltage record throughout, with much low-voltage, fast activity of about 30 cycles per second. There are rather frequent low-amplitude, slow potentials of 4 to 6 cycles per second in the F-O, F-M, and to a lesser extent in the M-O leads.

Right (E. E.): A white male of 37 years with general paresis, grade C, with convulsions, received a total of 30 hours of artificial fever therapy with 1.02 gms. mapharsen and 2.21 gms. bismuth. The blood remained positive, and the spinal fluid remained negative. The result was classed as a C. The electroencephalogram, taken four months later, showed an extremely low-voltage record. In the F-O leads, there are occasional outbursts of 7 cycles per second activity and low-amplitude, slow potentials of about 4 cycles per second from both hemispheres. In the M-O leads, there is activity of 10 cycles per second of irregular appearance and amplitude from the two hemispheres.

Figure 5, left (A. M.): A white male of 50 with general paresis, grade B, had received one year previously a total of 50 hours of artificial fever therapy combined with mapharsen and bismuth. There was only slight improvement, and he was kept on continuous chemotherapy for one year with no reversal of his blood or spinal fluid serology. Immediately before the second course of artificial fever, the electroencephalogram showed an extremely low-voltage record with much low-voltage activity of 24 cycles per second. There were occasional low-amplitude, slow potentials on the right in both the F-O and F-M leads.

Right (A. M.): A second course of artificial fever therapy, 30 hours, combined with mapharsen and bismuth, was given. The blood and spinal fluid serology was still positive. There was moderate clinical improvement. The electroencephalogram immedi-

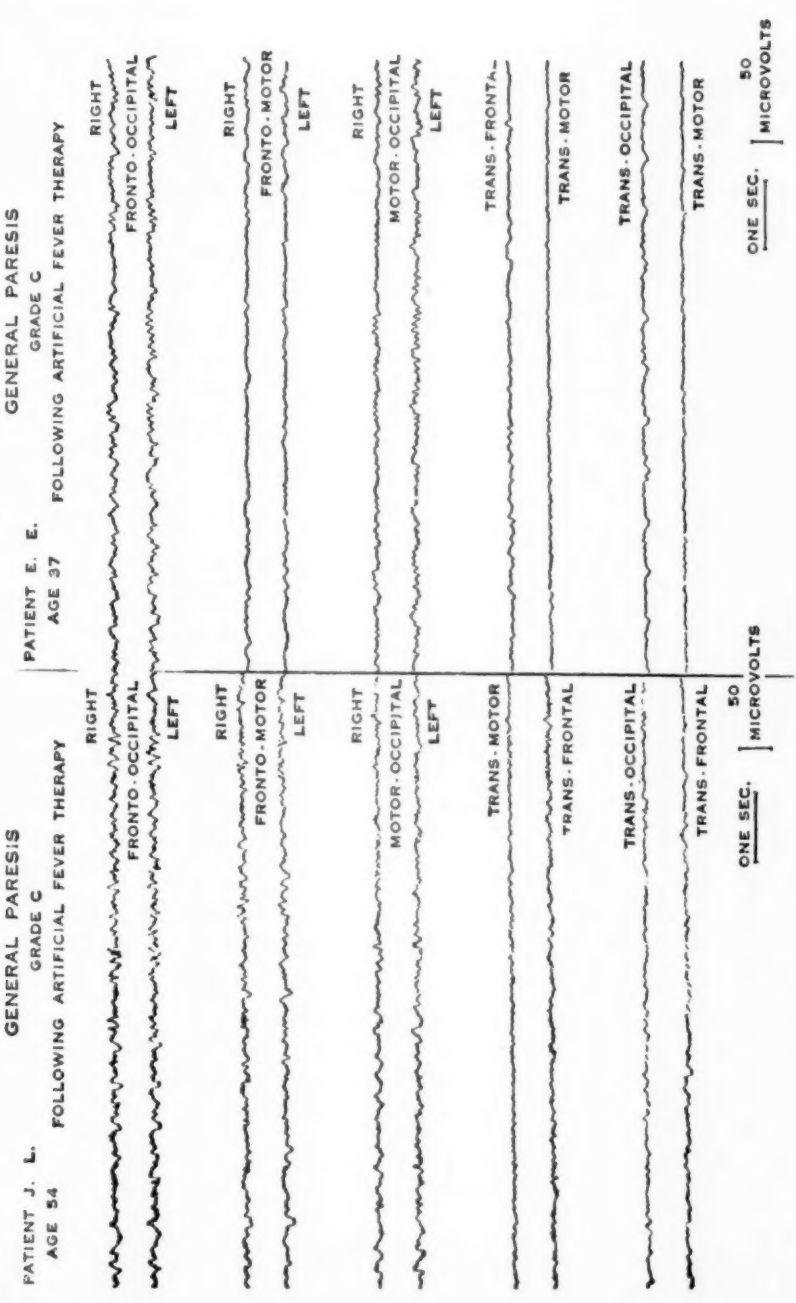

PATIENT J. L.
AGE 54
GENERAL PARESIS
GRADE C
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS-MOTOR
TRANS-FRONTAL
TRANS-OCCIPITAL
TRANS-FRONTAL
ONE SEC.
50 MICROVOLTS
PATIENT E. E.
AGE 37
GENERAL PARESIS
GRADE C
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS-FRONTA-
TRANS-MOTOR
TRANS-OCCIPITAL
TRANS-MOTOR
ONE SEC.
50 MICROVOLTS

Figure 4

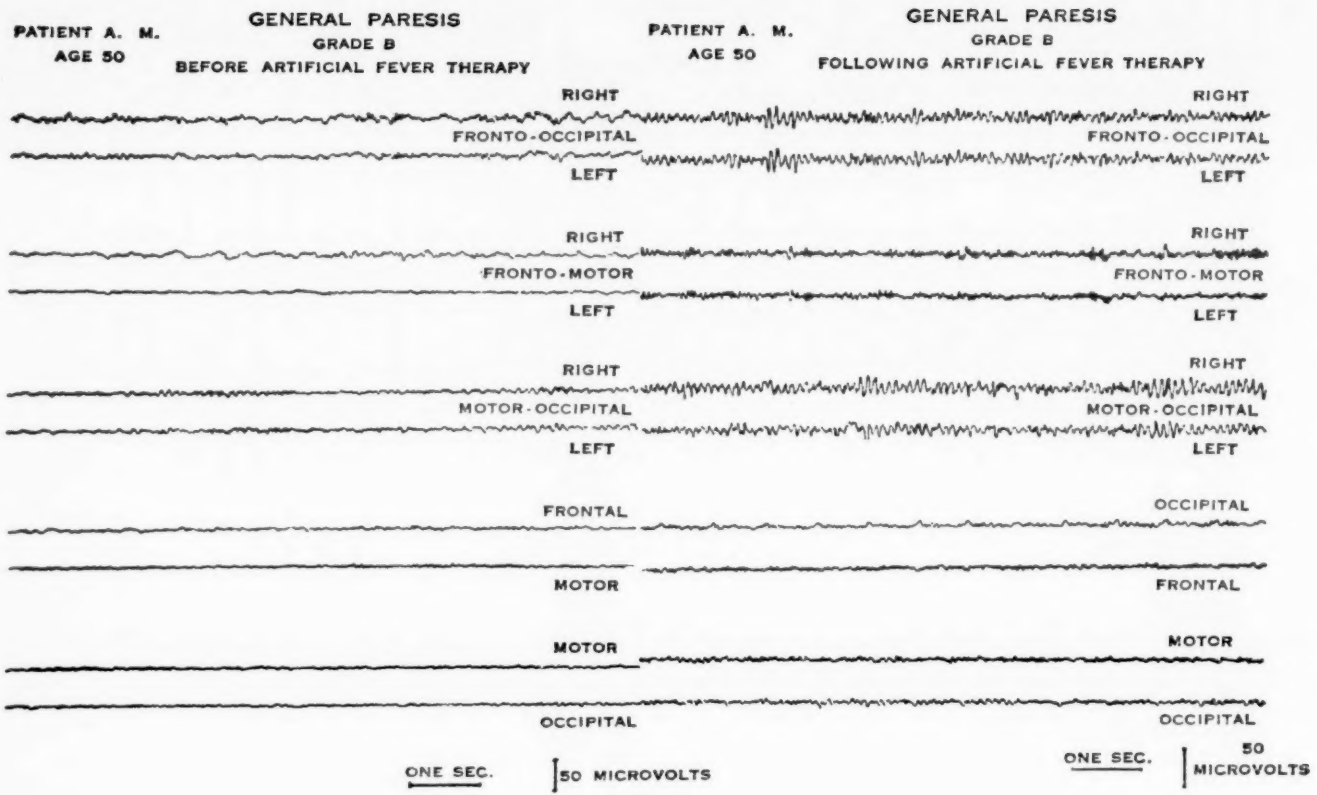

PATIENT A. M.
AGE 50
GENERAL PARESIS
GRADE B
BEFORE ARTIFICIAL FEVER THERAPY
PATIENT A. M.
AGE 50
GENERAL PARESIS
GRADE B
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
FRONTAL
MOTOR
MOTOR
OCCIPITAL
OCCIPITAL
FRONTAL
MOTOR
OCCIPITAL
ONE SEC.
50 MICROVOLTS
ONE SEC.
50 MICROVOLTS

Figure 5

ately after treatment showed a marked increase in amplitude with a return of alpha activity of 10 cycles per second in the F-O leads. The F-M leads show increased amplitude with the appearance of continuous fast activity of 26 cycles per second. There is a disappearance of the slow potentials.

Figure 6, left (C. F.): A white male of 51 years had general paresis, grade C, with convulsions. Blood and spinal fluid serology were strongly positive. The electroencephalogram shows a very low voltage record, marked by the appearance of slow potentials of about 3 cycles per second, most marked in the left frontal area but diffuse over the entire cortex. Only occasional brief outbursts of rhythmic activity of about 12 cycles per second occur. There is a definite asymmetry between the two hemispheres. In the transverse leads, the slow potentials are of greater incidence and amplitude.

Right (C. F.): This patient had 50 hours of artificial fever therapy, combined with aldarsone and bismuth. The blood and spinal fluid were unchanged at completion of the treatment. The result was classed as an A. The electroencephalogram showed a marked increase in amplitude, with a return of synchronous rhythmic activity of 11 cycles per second in the F-O and M-O leads. The F-O leads show a return of rhythmic activity of mixed alpha and beta waves. There is a disappearance of the slow potentials.

Figure 7, left (R. G.): A white male of 36, had had an unknown quantity of chemotherapy over a period of years. Paretic personality changes were mild, classed as grade A. The blood and spinal fluid serology was positive. The electroencephalogram showed the amplitude to be moderately depressed. There were frequent slow potentials of about 4 cycles per second in the F-O leads with only brief outbursts of rhythmic activity of varying amplitude and frequency. The F-M leads show a marked asymmetry, with a nearly normal appearance on the right and nearly continuous low-amplitude, slow potentials on the left. M-O leads showed nearly continuous slow potentials of 4 to 5 cycles per second with some superimposed low-voltage, fast activity. There were frequent slow potentials in all transverse leads.

Right (R. G.): After 24 hours of artificial fever therapy combined with aldarsone and bismuth the patient showed definite clini-

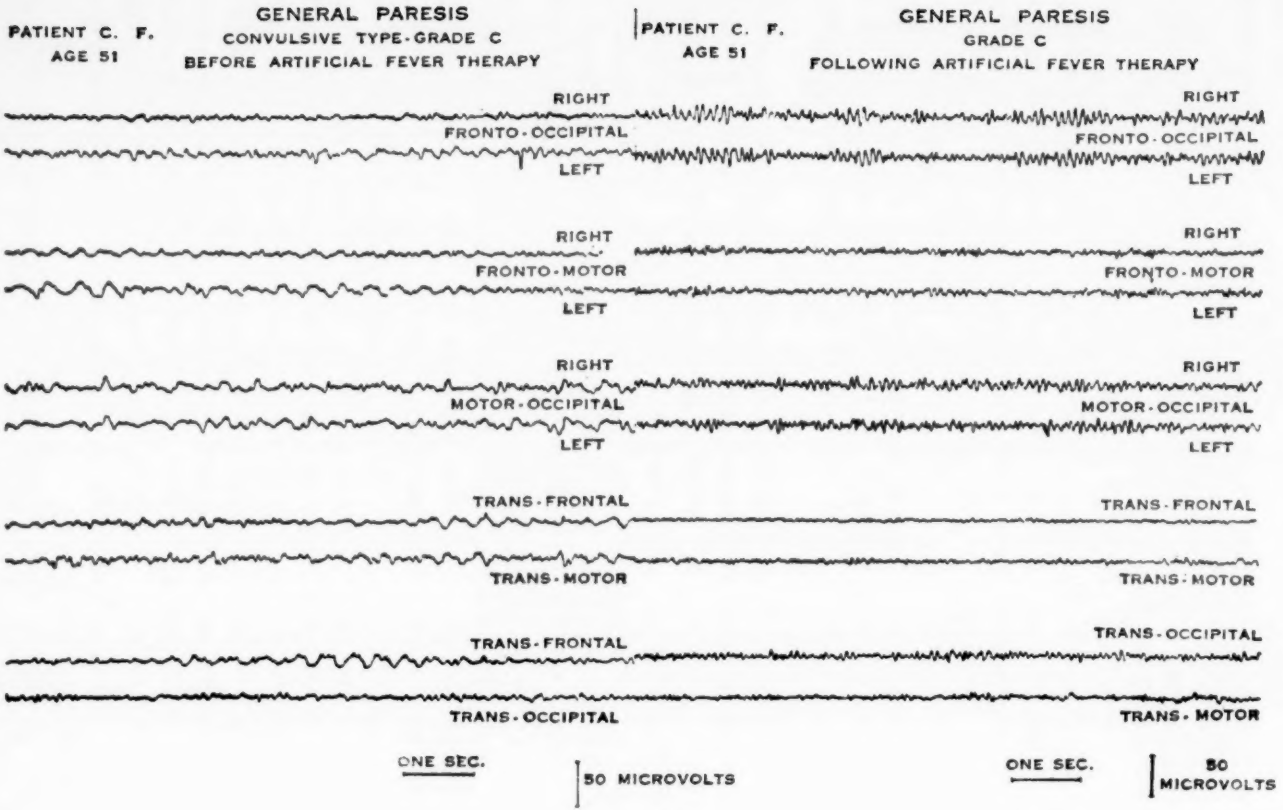

PATIENT C. F.
AGE 51
GENERAL PARESIS
CONVULSIVE TYPE-GRADE C
BEFORE ARTIFICIAL FEVER THERAPY
PATIENT C. F.
AGE 51
GENERAL PARESIS
GRADE C
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
TRANS-FRONTAL
TRANS-MOTOR
TRANS-FRONTAL
TRANS-MOTOR
TRANS-FRONTAL
TRANS-OCCIPITAL
TRANS-OCCIPITAL
TRANS-MOTOR
ONE SEC.
50 MICROVOLTS
ONE SEC.
50 MICROVOLTS

Figure 6

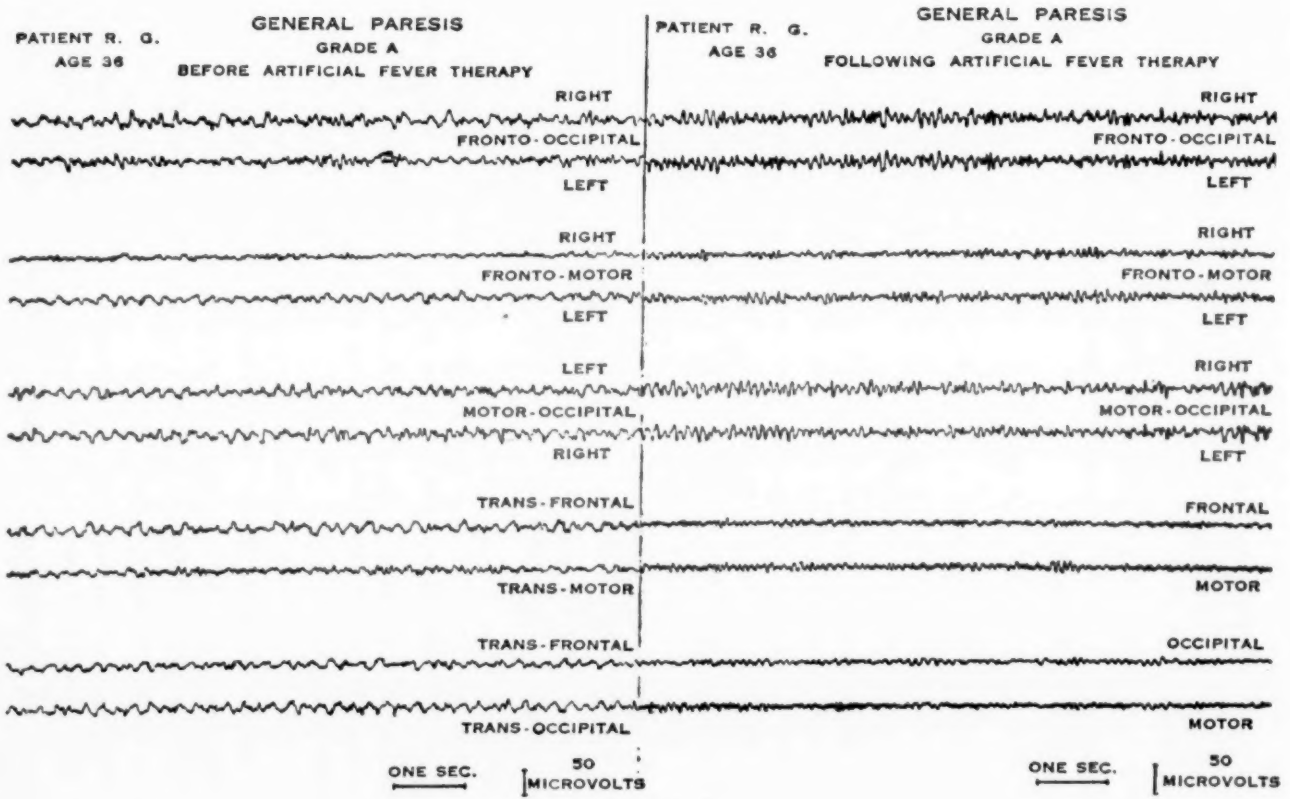

PATIENT R. G.
AGE 36
GENERAL PARESIS
GRADE A
BEFORE ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
LEFT
MOTOR-OCCIPITAL
RIGHT
TRANS-FRONTAL
TRANS-MOTOR
TRANS-FRONTAL
TRANS-OCCIPITAL
ONE SEC.
50 MICROVOLTS
PATIENT R. G.
AGE 36
GENERAL PARESIS
GRADE A
FOLLOWING ARTIFICIAL FEVER THERAPY
RIGHT
FRONTO-OCCIPITAL
LEFT
RIGHT
FRONTO-MOTOR
LEFT
RIGHT
MOTOR-OCCIPITAL
LEFT
FRONTAL
MOTOR
OCCIPITAL
MOTOR
ONE SEC.
50 MICROVOLTS

Figure 7

cal improvement. He did not return for treatment or a serology check but an electroencephalogram taken following his last treatment showed a definite increase in amplitude with return of rhythmic activity of about 10 cycles per second, but of varying frequency, amplitude and form. The slow potentials had completely disappeared; and there was a more symmetrical, synchronous appearance to the record.

Summary

The cumulative experience of workers treating general paresis with artificial fever and chemotherapy indicates results superior to those with malarial therapy.

In a series of 50 private cases, followed for three years (of which 82 per cent were of the mild or intermediate type), 56 per cent obtained full remissions, 90 per cent were improved, and 4 per cent died. Serologic reversals in the group were 34 per cent negative bloods and 63 per cent completely negative spinal fluids.

In a series of 79 state hospital cases of general paresis (of which 52.2 per cent were severe types), 19 per cent had full remissions, 32.9 per cent were further improved, 40.7 per cent were unimproved, and 7.9 per cent died. Serologic reversals in this group were 15 per cent negative bloods and 42 per cent completely negative spinal fluids.

Of these patients, 24 had had previous malarial treatment without obtaining full remissions, 13 were graded mild or intermediate types, 11 were severe. From this group, after artificial fever and chemotherapy, 29 per cent obtained full remissions, and 42 per cent were not improved.

In 15 cases of asymptomatic neurosyphilis with an average of 12 years infection, practically all resistant to previous vigorous chemotherapy, the bloods were completely reversed in 46.6 per cent and the spinal fluids in 73.3 per cent, while only 6.6 per cent failed to show serologic improvement.

In four cases of early acute general paresis, the electroencephalogram was abnormal and was distinguished by diffuse marked involvement of the alpha rhythm and the appearance of many slow potentials.

Three cases studied before and after treatment, in which clinical remissions were obtained, showed return to near-normal electroencephalograms immediately following treatment.

In a series of 10 consecutive cases which failed to obtain remissions, eight showed definitely abnormal electroencephalograms and one a borderline normal, after one to three years following treatment.

In seven of the 15 cases studied, the disturbance of electrocortical function appeared to be most marked in the frontal leads.

Departments of Fever Therapy Research and Psychiatry
University of Nebraska College of Medicine
Bishop Clarkson Memorial Hospital
Omaha, Nebr.

REFERENCES

1. Bessemans, A.: New Experimental Data on Hyperpyrexia. Trans. First International Fever Therapy Conference, New York City, 1937.
2. Schamberg, J. F., and Rule, Anna M.: Studies in the therapeutic effect of fever in experimental rabbit syphilis. Arch. Dermat. and Syph., 14:243, September, 1926.
3. Simpson, W. M.: Artificial fever of syphilis. J. A. M. A., 105:2132, December 28, 1935.
4. Boak, R., Carpenter, C. J., and Warren, S. L.: Studies on physiological effects of fever temperatures—The thermal death time of treponema pallidum vitro with special reference to fever temperatures. J. Exper. Med., 56:741, November, 1932.
5. Mehrtens, H. G., and Pouppirt, P. S.: Hyperpyrexia produced by baths. Arch. Neurol. and Psychiat., 22:700-709, 1929.
6. Neymann, C. A., and Osborne, S. L.: The treatment of dementia paralytica. J. A. M. A., 96:7-11, January 3, 1931.
7. Neymann, C. A.: Artificial Fever. Pp. 129. Charles C. Thomas Pub., Baltimore, 1938.
8. O'Leary, P. A.: The treatment of neurosyphilis. Proc. Mayo Clinic, 12:712, No. 12, 1937.
9. O'Leary, P. A., Bruetsch, W. L., Ebaugh, F. G., Simpson, W. M., Solomon, H. C., and Warren, S. L.: Malaria and artificial fever in the treatment of paresis. J. A. M. A., 115:677-681, 1940.
10. Jauregg, J. Wagner: The present management of progressive paralysis. Wiener Klinische Wochenschrift, 52:1075, 1939.
11. Berger, H.: Über das Elektrenkephalogram des Menschen. III. Arch. f. Psychiat. u. Nerv., 94:16-61, 1931.
12. Berger, H.: Ibid, VI. Arch. f. Psychiat. u. Nerv., 99:555-574, 1933.
13. Berger, H.: Ibid, VII. Arch. f. Psychiat. u. Nerv., 100:301-321, 1933.
14. Berger, H.: Ibid, XI. Arch. f. Psychiat. u. Nerv., 104:678-689, 1936.
15. Hoagland, H.: Pacemakers of human brain waves in normals and in general paretics. Am. J. Physiol., 116:604-615, 1936.
16. Finley, K., Gibbs, F. A., and Gibbs, E. L.: Atlas of Electroencephalography. P. 118. Lew A. Cummings Co., Cambridge, Mass.

RESULTS OBTAINED FROM THE ADMINISTRATION OF 12,000 DOSES OF METRAZOL TO MENTAL PATIENTS

A Preliminary Report

BY MARK ZEIFERT, M. D.*

Between October 30, 1937, and March 15, 1941, there were administered 12,005 metrazol treatments to male patients at Brooklyn State Hospital. Among the cases treated there were all types of schizophrenia, manic-depressive psychosis, involutional psychosis and the psychoneuroses. Eleven thousand, four hundred and sixty-nine treatments resulted in grand mal reactions, 415 resulted in petit mal reactions; and 70 were followed by psychic equivalents. Fifty-one treatments failed to produce any noticeable reaction.

With a few modifications, the technique used was essentially that originally described by von Meduna. In place of the aqueous solution of metrazol which was used for the first 3,000 treatments, a 10 per cent solution of metrazol in 2.5 per cent sodium citrate was substituted. The anticoagulant was added to prevent formation of blood clots in the syringe after penetration of the vein. In addition to this, since October, 1939, calcium gluconate and cod liver oil have been administered *per os,* in a procedure based on the observations of Dr. C. H. Bellinger on both rabbits and humans, that a great many subjects show radiographic evidence of bony decalcification during metrazol therapy. Finally, the patients have been placed in the position of hyperextension of the upper spine during treatment. The latter two precautions have been taken to prevent the occurrence of compression-fractures of the anterior borders of the thoracic vertebral bodies, and the incidence of such injuries has thus been reduced to a great degree.

The youngest patient in this series was 16, while the oldest was 57. The average weight of patients before therapy was 147 pounds, while after treatment it was 149.5 pounds, a gain of 2.5 pounds.

Among the patients who received treatment but failed to reach a stage that permitted discharge, were noted a considerable number who improved in conduct. Many who soiled and wet themselves

*Since this paper was written, Dr. Zeifert has been called to active duty as a captain in the United States Army Medical Corps. He is stationed at Camp Blanding, Fla.

began to take care of their own hygienic needs. A number who were violent and destructive prior to treatment improved in conduct, notwithstanding the fact that they retained their delusional trends.

Certain complications have been observed among the Brooklyn State Hospital patients. Prior to the time the use of citrated metrazol solution was begun, there were seven cases of pulmonary infarction (the so-called "lung abscess" of other writers) in 3,000 treatments. In the next 9,000 treatments, no cases of infarction or "lung abscess" developed, which is attributed at Brooklyn State Hospital to the presence of anticoagulant in the solution. Subluxation of the mandible was a frequent occurrence early in experience at this hospital; but it was soon learned how to prevent it—by instructing the attendant to insert the mouth-gag into the patient's mouth as soon as the convulsive yawn appeared, and then to cup his hands around the patient's lower jaw and press upward in such a manner as to limit the excursion of the jaw.

One patient developed status epilepticus following the injection of two doses of metrazol in the amount of 5 cc. each. The first injection having failed to induce a seizure, a second was given two minutes later. He immediately had a convulsion which was followed by a series of them. Intravenous injection of 7½ gr. of sodium amytal resulted in complete cessation of the epileptoid phenomena.

There was one case of subluxation of the shoulder, but this was easily reduced under nitrous oxide anaesthesia.

One patient sustained a cortical fracture of the head of the radius. Another patient suffered a fracture of the neck of the humerus, while in still another patient there was a fracture of the body of the scapula.

During the Brooklyn State Hospital's first month's experience with metrazol treatment, there were two fractures of the neck of the femur. The impression was gained that the fractures of the long bones are due either to manual or mechanical restraint and that free play should be given to the lower extremities during the seizure, while the arms should be kept in the direction of adduction.

There were a number of compressions of the anterior borders of the thoracic vertebrae prior to the time the administration of cal-

cium gluconate and cod liver oil was begun and before the practice was started of inducing the seizures while the patient was in a state of vertebral hyperextension. These compressions occurred almost exclusively, as has been reported by many writers, in the mid-thoracic vertebrae. In every case these injuries were asymptomatic; and although some patients complained of pain in the back, this symptom soon disappeared and was not replaced by others. At no time was it possible to demonstrate evidence, either subjective or objective, of peripheral nerve or spinal cord involvement. X-rays of these spines were extremely interesting and Dr. Bellinger, the superintendent of the hospital, in conjunction with the consulting roentgenologists and orthopedists, made a special study of these plates. The bodies of these vertebrae, apart from the wedging of the anterior borders, presented a peculiar moth-eaten appearance and relative radio-lucency which was interpreted as evidence of demineralization. The borders of many of the vertebrae presented a concave appearance described by roentgenologists as a fish-mouth deformity. The degree of decalcification was greater in the young patients than in the older ones, which in all probability could be accounted for by the fact that bone calcium is more firmly fixed as individuals advance in age. In these cases, some of the other bones, notably the upper portions of the femora, also showed demineralization—in some instances, cystic in appearance. On the advice of the consulting orthopedists, these injuries were treated by the administration of cod liver oil, calcium gluconate and physiotherapy in the form of ultraviolet rays.

There were no deaths during convulsion, nor immediately following it. However, four patients in the series died. One patient developed pneumonia one day after treatment and died five days later. The second patient died of pulmonary infarction. Another died three weeks after his last metrazol treatment, following operation for open reduction, under ether anesthesia, of a fracture of the femoral neck sustained during a convulsion. The fourth patient, diagnosed as dementia præcox, paranoid type, had been ill for 52 months. Metrazol therapy was begun June 6, 1940, and continued until August 27, 1940. Although the patient had reacted on each occasion with a grand mal seizure and had experienced 25 such convulsions, he failed to react in typical fashion on the last day.

Instead, he developed a psychic equivalent with wild excitement. He was extremely powerful and, even though 15 grains of sodium amytal were administered intravenously, he continued to struggle in a manner similar to that seen in Bell's mania. The patient soon developed a state of exhaustion with vasomotor collapse and failure of the thermo-regulatory center. Notwithstanding active treatment, he developed a temperature of 109 degrees and died three hours after the administration of the metrazol.

Analysis of Results

Of 19 cases of manic-depressive psychosis treated, 18 improved to the extent that they were able to leave the hospital; and notwithstanding the fact that many of them have been discharged for months, none has shown a return of mental symptoms.

Four patients with involutional melancholia treated with metrazol were all released. Of five with the paranoid type of involutional psychosis, four were released and one failed to improve. There were no returns to the hospital in the involutional group.

Three cases of psychoneurosis were treated, of which number two recovered, while one failed to improve. Both of the patients who recovered are now gainfully employed and free from symptoms.

In the schizophrenic group, 341 patients completed the course of treatment. Of these, 147 were released either on parole or by discharge. Twelve of these patients were returned to the hospital because of recurrence of the symptoms. The number released was 43.1 per cent of the total treated.

The number returned from parole was 8.1 per cent of the total number released. The table depicts the result of treatment in the schizophrenic group of patients.

Results of Treatment in Schizophrenic Group of Patients

Duration of illness	Number treated	Recovered	Much improved	Improved	Unimproved	Total released
1 to 6 months (acute)	64	16	26	18	4	54
7 to 24 months (intermediate) ..	125	13	25	45	42	59
25 months to 30 years (chronic)	152	4	11	43	94	34
Total	341	33	62	106	140	147

As has been pointed out by many other writers, there is a direct relationship between duration of illness and prognosis. This is clearly evident in the first group of cases, ill less than six months, whose parole rate was 84.3 per cent. In the intermediate group, where the duration of illness was seven to 24 months, the parole rate was 47.2 per cent. The last group, consisting essentially of chronic cases (duration 25 months to 30 years), had a parole rate of only 22.3 per cent.

A study of this table reveals certain other information of great value. For example, it appears that, regardless of the duration of illness, the chances for "complete recovery" are not good. Out of the 201 patients who benefited from the treatment, only 33 "recovered," while the remainder were classified as either "much improved" or "improved." It is also evident that where the treatment is efficacious, there is a tendency for more complete adjustment in the acute cases than in the intermediate or chronic group. This is shown by the fact that the ratio of "recovered" and "much improved" patients to those only "improved" in the acute group was 42 to 18. In the intermediate and chronic groups, the ratio was only 38 to 45 and 4 to 54, respectively.

Further study of this table leads to the belief that patients with early cases either do not enter the hospital for treatment, or that their relatives are reluctant to give consent for treatment. Notwithstanding the fact that no attempt was made to select patients for treatment, there were only 64 acute cases as against 125 intermediate and 152 chronic cases in this series.

Discussion

Metrazol therapy has proven to be a very valuable agent in the treatment of mental patients in Brooklyn State Hospital. The staff is aware of the fact that the results here are more hopeful than those reported in some other places. It is difficult to explain why this should be, for notwithstanding local differences of opinion regarding some of the finer points, the concept of mental disease is essentially the same throughout the United States. The writer is of the opinion that it is more accurate to determine the value of chemotherapy on the basis of parole results than on degrees of improvement. Opinions differ widely as to degrees of remission, but

the criteria for returning patients to community life are practically the same in all instances. Therefore, it would seem to the writer that statistics based on parole records offer the most accurate basis for comparison of the results obtained in the various hospitals.

The course of treatment at this hospital has invariably been longer than that given at some other institutions. This, it is believed here, accounts for the results obtained with many moderately advanced cases. In the involutional psychoses, from 12 to 20 treatments were administered, as a rule. In all cases, treatment was continued until the patient gave evidence of confusion which, it is the opinion at Brooklyn State Hospital, is highly important and should constitute the end-point. It has been the experience here that people in the later decades of life reach this state with fewer injections than younger patients; and this probably explains why the involutional cases responded to fewer treatments than did those of the manic-depressive or schizophrenic groups. The latter required more treatments than did any other type; and it was at times necessary to administer more than 30 injections at the usual rate of three a week. Here again, the physicians found themselves obliged to carry the therapy to the point of confusion rather than to adhere to any fixed number of treatments. In this form of therapy, as in all other psychotherapeutic measures, the patient should be treated as an individual, with no hard and fast rule laid down in advance. The individual should be observed from treatment to treatment; and termination of therapy must be only on the basis of the patient's reaction at the time, rather than on any predetermined plan. To illustrate this, the writer calls attention to two patients with catatonic dementia præcox, both in the fourth decade of life, one of whom showed a splendid remission after the twelfth treatment while the other presented no evidence of improvement until the thirty-eighth and did not achieve full remission until after the forty-sixth convulsion. There is, of course, no fixed guide to the number of treatments necessary; and at times it becomes a matter of fine clinical judgment to determine whether it is worth while continuing beyond a certain number of convulsions. The more delicate the clinical sense of the physician, the higher is the percentage of paroles.

The writer has previously called attention to the importance of continuing treatment for some time after remission begins to appear; and this observation has been confirmed by continued experience. Unless three to six convulsions are induced after remission first makes it appearance, the confusion is too transient. It requires from three to six weeks for this confusion to disappear; and no patient should be permitted to leave the hospital until his sensorium is perfectly clear. It is far better for the patient to remain under observation for a month or six weeks than it is for him to be released too early and risk the possibility of return from parole because of difficulties engendered by the inability of a confused person to adjust satisfactorily at home. The writer feels that in the Brooklyn State Hospital series the low percentage of patients returned from parole is due to the fact that they had an opportunity to adjust in the carefully-regulated environment of the hospital. Those who failed to do so were not paroled.

Conclusions

1. The results obtained from 12,000 convulsions induced in male patients by the administration of metrazol have been analyzed.

2. Complications and methods of prevention have been discussed.

3. Metrazol is most efficacious in the treatment of involutional and manic-depressive psychosis regardless of duration of illness.

4. In schizophrenia, the rate of remission is in direct proportion to the duration of illness.

5. The results here obtained warrant further therapeutic use of this modality.

Brooklyn State Hospital
Brooklyn, N. Y.

THE RELATION OF A GROUP OF HIGHLY-IMPROVED SCHIZOPHRENIC PATIENTS TO ONE GROUP OF COMPLETELY-RECOVERED AND ANOTHER GROUP OF DETERIORATED PATIENTS

BY OTTO KANT, M. D.

In a previous study[1] the general characteristics and the clinical pictures of two comparable groups of 39 completely-recovered and 39 deteriorated schizophrenic patients were compared. The chief results of this investigation will be summarized later in this paper. It may suffice here to say that definite differences in the general characteristics as well as in the types of clinical pictures could be demonstrated. Since the results obtained concern only the two prognostically extreme groups of schizophrenia, they are so far of limited value. It is the purpose of the present study to compare the previous findings with those in a group of schizophrenic patients who are intermediate between those in the "completely-recovered" and those in the "deteriorated" groups. By this means, the scope of the characteristics pointed out for the end groups will be examined. The question to be decided is whether the differentiating features are limited exclusively to the end groups or, if not, to what degree they can be recognized in the intermediate group.

The group examined in the present study consists of 22 patients* who had received routine medical and psychiatric care, but no kind of shock or prolonged narcosis treatment. All of these patients, the majority of whom have been out of the hospital for more than five years, were personally reexamined by the writer and at that time considered as highly improved.

The definition of "highly improved" used here is rather broad. It covers all those patients who have resumed work at about the same level as before their illness and who have been able to adjust, at least to a fair degree, to their old environments. All of these patients, however, exhibited some traits in their general attitude

*The group of 22 highly-improved patients was collected in connection with a survey of completely-recovered schizophrenic patients.[2] Because of the small number of patients in this group, a comparison with the two extreme groups statistically may not be conclusive. The analysis of the material, however, indicated such a number of marked common features that it appeared justifiable to examine the relation of this group to the two extreme groups.

or behavior which prevented the examiner from considering them as completely recovered. The abnormalities represented in this group are not all of the same type. In more than half of the patients (12) there were at least slight signs of emotional deterioration (loss of vigor, apathy) or of some other permanent change in the emotional sphere, for example, chronic hypomanic attitude. The remaining 10 patients did not actually appear to be deteriorated at all; their defects consisted of lack of true insight into their previous psychoses, or of some eccentricity of behavior, or of the presence of slight residual symptoms, such as occasional hallucinations or slight blurring of thought. These three types of defects appeared either alone or in various combinations, and in some patients were associated also with slight emotional blunting. In the numerical evaluation of general features in the highly-improved group, no differentiation is made among these various types of abnormalities, since the primary purpose of this study is to ascertain the general characteristics of all highly-improved patients, whatever their deviations may consist of.

Because of the relatively small number of patients in this group, some of the characteristics mentioned in the writer's previous studies have not been used here. Only those items which have proved to be of differentiating importance for the two extreme groups are considered.

Results

Table 1. Per Cent Distribution of Five Outstanding Features in a Recovered Group, a Highly Improved Group, and a Deteriorated Group

Feature	Percentage in recovered group	Percentage in highly-improved group	Percentage* in deteriorated group
Psychogenic precipitation	74	50	15
Clouding of consciousness....	67	41	8
Acute or subacute onset.....	85	23	18
Extraversion	72	54	8
Pyknic physique	51	50	10
Mean:	70	44	12
Proportion:	6 :	4 :	1

*It may not be statistically justifiable to compute percentages of such a small number of cases; but for purposes of comparison, this method seems rather advantageous.

The selection of the five features considered in this table was originally determined by their importance in the recovered group. As is shown clearly, the highly-improved patients in their general characteristics are much more closely related to the completely recovered than to the deteriorated group. While the proportion is 1:4 between the deteriorated and the highly-improved groups, it is only 1:1.5 between the latter and the completely-recovered group.

The closest approach of two figures is seen in the distribution of pyknic physique, which is 50 in the highly-improved and 51 in the recovered group. The greatest divergence in figures between these two groups occurs in the type of onset. The fact that there is a larger proportion of psychoses of a depressive type in the highly-improved group (3:2) can only partly account for this difference; acute or subacute onset, which occurred in 85 per cent of the recovered group, was reported in only 23 per cent of the highly-improved group.

The proportion of the combination of extravert temperament and pyknic physique is not much smaller in the highly-improved than in the recovered group (41 to 44 per cent); in the writer's previous investigation, this combination was not represented at all in the deteriorated group.

The combination of introvert temperament and either leptosomic, athletic, or dysplastic physique, which was found in 3 per cent of the recovered group and in 33 per cent of the deteriorated group, is found in 9 per cent of the highly-improved group.

Some of those psychopathic features (emotional lability, neurotic attitudes, lack of maturity) which seemed to be quite as important for the personality type of the recovered group as extraversion (70 per cent) were to a smaller degree represented in the highly-improved group (40 per cent). As pointed out previously, these psychopathic features were difficult to appraise in the deteriorated group, where they may have largely been covered over by the predominant introvert attitude. At the same time, it had been mentioned that about one-fourth of all patients in the deteriorated group appeared to have been "queer" or "nervous" from early childhood; this seems to hold true with nearly one-fifth of the patients in the highly-improved group, while none of the patients in the recovered group seemed to fall under these headings.

Number of Attacks

There were more than one psychotic attack (from two to nine) in 55 per cent of the highly-improved patients as compared to 36 per cent in the recovered and 30 per cent in the deteriorated groups.

Of those 12 patients of the highly-improved group who had more than one attack, nine seemed to give the same clinical impression at the end of their first attack as they do at present. In three cases, however, the patients appeared to be actually recovered after the first and/or the second attack, while there remained some permanent abnormalities after a later attack. Because of their importance, brief abstracts of these three cases are given.

In the first case, that of a female patient apparently recovered after her first attack, the clinical picture was that of a paranoid depression precipitated by childbirth. The second attack, after which a slightly paranoid attitude seems to have remained, is best characterized as a paranoid-excitement state with some clouding of consciousness during the earlier part of the psychosis.

In the second case, a male patient, who so far has had six attacks, exhibited the clinical picture of an agitated depression with anxiety during the first two periods. From the third attack on, paranoid trends were more predominant in the clinical picture. Since this third attack, the patient seems to have remained nervous and irritable and occasionally to have had spells with minor paranoid reactions.

In the third case, a female patient appeared to be recovered after her first two psychotic attacks in which some clouding of consciousness was an important feature. Both attacks, which originally were diagnosed as "delirium" and "atypical depression," were precipitated by the puerperium. In all later attacks, clouding was either absent or a minor feature; and the patient, who in the following periods impressed the observers chiefly by her manic behavior, later developed some schizophrenic trends also (grimacing, paranoid experiences, etc.) At present, she shows slight signs of emotional deterioration and of "queer" behavior as well as some lack of insight.

It is noteworthy that the clinical pictures of all three patients showed some change also after the first or second illness.

Characteristics of the Clinical Pictures in the Highly-Improved Group

In more than half of the total number of cases (12 of 22) the psychoses showed some strong manic-depressive admixtures, herewith distinctly reminding one of the recovered group.

It has already been mentioned that 10 of the highly-improved patients showed no signs of emotional deterioration or of any other permanent change in the emotional sphere. Since permanent emotional changes are still regarded as the chief sequelae of the schizophrenic process, it was thought advisable to discuss the clinical pictures of the highly-improved group in two subdivisions: (a) those patients in whom these changes were not present, and (b) the remaining patients.

(a) In the clinical pictures of all patients in this subgroup, some kind of depressive changes or of excitement states were predominant. Of 10 patients, eight resembled to a greater or less degree those clinical types which have previously been described[2] as characteristic for the recovered group. To approach the question of differentiation, the short summary, given in one of the writer's preceding papers,[1] of the description of the five clinical types which have been established, is quoted here:

"Although all of these might conventionally be diagnosed as schizophrenic, the classification has been made in the order of prevalence of atypical features. These are most prominent in the first and least evident in the last group:

"Group I: Resembling atypical depressive states.

"Group II: Resembling atypical manic states.

"Group III: Manic and depressive features outstanding.

"Group IV: Alternating conditions of excitement and stupor with some manic-depressive trends.

"Group V: Prevalence of schizophrenic symptomatology.

"While the first three of these groups show a close relation to the manic-depressive psychosis, Group IV stands by itself. Excitement and stupor are predominant, but true autism and other signs of schizophrenic dissociation still are missing. Group V finally most closely approximates schizophrenia in its ordinary

conception. There is, however, one feature which is remarkable through this whole group: that is, the psychogenic precipitation and the psychogenic coloring of the psychotic attack."

Two patients in (a) exhibited clinical pictures which cannot be differentiated from those in Group I of the recovered group. The remaining abnormalities of these two patients (lack of objectivity toward the previous psychosis in one, and some eccentric behavior in the other) are probably due to their personality makeup.

The clinical pictures of four patients greatly resembled those described in Groups I, III, and IV respectively; but in each case, there were some slight differences in the structure of the psychosis (for example, absence of clouding or of psychogenic precipitation) which may account for the fact that the prognosis was not quite so good as in the completely recovered patients.

In three cases, the pictures exhibited features which to some degree reminded one of those in Group I as well as in Group V, without permitting their inclusion in any single group. On the whole, however, there were in these cases many of those features which had been particularly recognizable within the recovered groups (clouding, psychogenic precipitation, uniformity of the abnormal content, and lack of bizarre characteristics).

The one remaining case can be characterized as a psychogenically precipitated paranoid reaction of a patient of borderline intelligence who still appeared inclined to have some abnormal experiences.

(b) Two of the patients in this subgroup, in which there is somewhat more evidence of chronic schizophrenic changes than in (a), exhibited clinical pictures to some degree suggesting those in Group I of the recovered group, similar to those mentioned in (a). While the first two attacks of one of these patients appeared to be chiefly of a depressive character, later some change of the general coloring occurred in which paranoid experiences prevailed.

The psychoses of six other patients are best characterized as "mixed" or "schizo-affective" psychoses, since their symptomatology consists of manic-depressive trends as well as of schizophrenic features. In spite of the strong manic-depressive admixtures, none of these clinical pictures resembles those structures described as characteristic for the recovered groups. While the

schizophrenic symptomatology in the latter occurred chiefly in the special setting of clouding or of incoherence and mostly seemed to correspond to the affective changes, this was not the case in the mixed psychoses mentioned here. Manic-depressive and schizophrenic changes occur either combined with or following each other, each of equal importance within the setting. The schizophrenic trends in this group consist of lack of affective response, autism, grimacing, and of paranoid delusions. Two of the six patients suggest the so-called paraphrenic type. Both of them, who were over 44 when the attack started, now combine hypomanic extraversion with a completely uncorrected and somewhat fantastic attitude toward their previous abnormal experiences.

The question whether the "mixed psychoses" in the highly-improved group can from the beginning be clinically differentiated from those schizophrenias which lead to deterioration in spite of manic-depressive admixtures cannot be definitely answered on the basis of the present case-material. The writer has the impression, however, that there are more bizarre features and other signs of personality disintegration in the malignant cases. It is remarkable that five of the six patients with "mixed psychoses" are definitely extraverted and show a predominantly pyknic physique.

There remain to be discussed four patients whose psychoses neither can be described as resembling those in the recovered group nor characterized as "mixed psychoses."

Two patients have, in common, psychogenic precipitation by a frustrated love affair. The abnormal content of their psychoses remains centered around these experiences; but there is no clouding of consciousness, and definite schizophrenic signs (for example, feeling of foreign influence in one and discordancy of behavior in the other case) are present. The third patient is of borderline intelligence. Psychogenic factors seem to have been of some importance in the precipitation of at least the first two of his four psychotic attacks. In character these are alternately more hebephrenic and more hebephrenic-paranoid.

The fourth patient is a male whose gradually beginning paranoid psychosis was chiefly characterized by feelings of foreign influence and auditory hallucinations; that is, the patient felt that he was forced to do things, that voices controlled him, that they made use

of his vocal cords and governed his conduct. During the entire psychosis, he was "in excellent contact" with his surroundings. In spite of the predominant experiences of foreign influence, there were no other signs of personality dissociation or disorganization. The patient now appears well composed, but does not seem to have regained true insight for his abnormal experiences. He appears slightly blunted emotionally and has somewhat Utopian plans for his future.

Comments and Conclusions

The fact that the general characteristics of the highly-improved group are intermediate between those of the recovered and the deteriorated groups argues for the broad scope of their importance. There is a distinct correspondence between the numerical proportion of certain outstanding features and the clinical outcome in each group. One may, therefore, draw the conclusion that the mentioned characteristics are a differentiating aid for all prognostic varieties of the schizophrenia group. The greater the number of certain features present, the greater is the probability that the prognosis is in the direction of recovery. It must be recalled, however, that the proportions mentioned hold true for each group as a whole and that they cannot be considered representative of each individual case. The criteria are thus mainly of value in indicating the general direction of the development.

On the whole, the types of clinical pictures in the highly-improved group were much more closely related to those of the recovered than of the deteriorated group. This again suggests that the clinical pictures described in the recovered group are types which are most characteristic for the benign cases of schizophrenia, but show manifold transitions in intermediate cases. From a general point of view, manic-depressive admixtures were most characteristic for the highly-improved as well as for the recovered group. This confirms the general results of Mauz[4] and many later investigators, including the most recent comprehensive studies of Langfeldt[5] and of Blair,[6] that the presence of manic-depressive features in a schizophrenic picture usually indicates at least a fair prognosis.

Those cases in the highly-improved group described as "mixed psychoses" can well be differentiated from those in the recovered group in which affective changes were predominant. Future investigations must clarify whether it is possible to make a reliable clinical differentiation of those "mixed psychoses" of the highly-improved group from the schizophrenias with manic-depressive admixtures which lead to deterioration.

In those cases of the highly-improved group in which the manic-depressive element was not remarkable, psychogenic features were of central importance—at least in two of three psychogenically-precipitated cases. The schizophrenic symptomatology, however, was so outstanding that the writer should not dare to differentiate these psychoses from others which had a much worse outcome. Finally, there is one case in which the clinical picture from the beginning was characterized by experiences of foreign influence without any other signs of personality disorganization. This patient apparently managed to maintain a well-composed personality in a paraphrenic manner by keeping his psychotic experiences somewhat at a distance from his personality nucleus. It will still remain to be determined whether this kind of setting is always of the same prognostic importance in cases where similar schizophrenic symptomatology is present.

The fact that in some individual cases it still remains doubtful whether the prognosis will be "moderately good" or "moderately bad" does not invalidate the general prognostic aid offered by the analysis of the clinical pictures in the highly-improved group as well as in the two extreme groups.

It is interesting to note that there were no true catatonics in this group of 22 highly-improved schizophrenic patients. It would, therefore, seem that the catatonics are destined either to recover completely or to deteriorate.

Summary

1. Twenty-two schizophrenic patients who had received no shock treatment, the majority of whom had been out of the hospital for more than five years, were at the time of personal reexamina-

tion found to be "highly improved." The results of this investigation were compared with those of studies of a group of completely recovered and a comparable group of deteriorated schizophrenic patients.

2. The general characteristics of the "highly-improved" group proved to be intermediate between those of the two extreme groups, but much closer to the recovered group, with the exception of the types of onset. In the predominance of gradual onset of the psychoses, the "highly-improved" group distinctly resembled the deteriorated group.

3. Most of the clinical pictures in the "highly-improved" group, like those in the recovered group, exhibited some kind of manic-depressive admixtures. Those cases in which permanent changes in the emotional sphere, as a result of the schizophrenic process, were not typical showed closer relation to certain types of clinical pictures described as characteristic for the recovered group than did the remaining cases. Of the latter, several might be characterized as "mixed" or schizo-affective psychoses, which differ distinctly in their structure from the clinical types in the recovered group. Future investigations are needed to determine whether a clinical differentiation from similar cases leading to a deterioration is also possible. Some features which might be of prognostic significance are pointed out.

As opposed to both extreme groups, the highly-improved group does not present any true catatonic pictures.

4. The results of this investigation supply a further indication for the differentiating importance of some general characteristics and certain types of clinical pictures within the schizophrenia group—factors which have been stressed throughout this series of studies.

Research Service
Worcester State Hospital
Worcester, Mass.

REFERENCES

1. Kant, O.: A comparative study of recovered and deteriorated schizophrenic patients. J. N. M. D., 93, 5, 616-624, May, 1941.
2. ——:Study of a group of recovered schizophrenic patients. PSYCHIAT. QUART., 15:2, 262-283, April, 1941.
3. ——: Types and analyses of the clinical pictures of recovered schizophrenics. PSYCHIAT. QUART., 14:4, 676, October, 1940.
4. Mauz, F.: Die Prognostik der endogenen Psychosen. 1930, Leipzig.
5. Langfeldt, G.: The prognosis in schizophrenia and the factors influencing the course of the disease. 1937, Copenhagen, London.
6. Blair, D.: Prognosis in schizophrenia. J. Ment. Sc., 86:378-477, No. 362, May, 1940.

MAPHARSEN IN THE TREATMENT OF THERAPEUTIC BENIGN TERTIAN MALARIA*

BY DUNCAN WHITEHEAD, M. D.,† AND JOHN J. DOREY, M. D.

The frequent difficulty in obtaining negative blood smears for the malarial parasite on three consecutive days and the often-encountered unfavorable toxic reactions produced by quinine led to the experimental adoption of an arsenical in the treatment and termination of therapeutically induced malaria. It was established that arsenicals have been successfully used in the therapy of malaria as evidenced by standard textbooks. Recently Goldman has reported on his use of mapharsen for this purpose with considerable enthusiasm. In the writers' experience with a small series of cases, control of therapeutic malaria in most of them has been obtained by the use of mapharsen with no discomfort to the patient. However, the writers' findings indicate that some precaution is necessary since mapharsen may not bring about complete eradication of parasites from the blood.

Literature

The use of arsenicals in malaria is by no means a new development. Stitt[1] in his standard text on tropical diseases in 1917, stated, while discussing the treatment of malaria: "Salvarsan and neosalvarsan have been extensively used and with some success in benign infections but without material effect in malignant tertian ones."

Some modern textbooks give no mention to the possible treatment of natural or therapeutically induced malaria by arsenicals. Among these are Cecil's "Textbook of Medicine,"[2] "Osler's Principles and Practice of Medicine,"[3] as edited by McCrae, and "Textbook of Nervous Diseases," by Bing and Haymaker.[4] Moore[5] in his, "The Modern Treatment of Syphilis," speaking of therapeutically induced malaria, states: "In the rare quinine-sensitive patients, the infection may be satisfactorily terminated with an in-

*Read at the up-State interhospital conference at Utica State Hospital, April 26, 1941.

†Since this paper was written, Dr. Whitehead has been called to active military service. He is a major in the United States Army Medical Corps, stationed at the Lovell General Hospital, Fort Devens, Mass.

jection or two of an arsphenamine." Henderson and Gillespie,[6] in "A Textbook of Psychiatry," speaking of the same subject, say, "In quinine-sensitive patients, an arsenical salt, e. g., neoarsphenamine, may be injected instead."

Beckman,[7] in "Treatment in General Practice," quotes at length from Muhlens (1932) concerning the use of arsphenamine in the treatment of natural malaria. Here the arsphenamines are recommended in the early treatment of malaria because they "abort the symptoms in practically every attack of malarial fever." In citing reasons for the utility of neoarsphenamine, the following are given: "(a) It stops the attacks promptly before the patient is greatly weakened by the fever and plasmodial toxins, (b) through its excellent tonic value, it rapidly builds up the patient, (c) patients recover their strength quickly and return to work considerably sooner than with quinine treatment alone, (d) it is effective when even quinine in enormous doses fails, i. e., where quinine-fast organisms are present, or for some reason the quinine is not properly assimilated." He further states that neoarsphenamine alone is specific in tertian malaria. The use of other arsenical preparations is also mentioned in the same connection.

In 1938, Goldman[8] reported on the use of mapharsen in the treatment of malaria, both natural and artifically induced. With therapeutic malaria he found that "in most cases a single injection suffices to terminate the malaria permanently, but to insure against recurrences it is well to give three or four injections at the proper intervals." He found relapses following a single dose in only two cases out of 24 treated. However, he advocated the use of eight to 10 injections for mapharsen's antisyphilitic value and, at the same time, for the permanent eradication of the malaria. He encountered no toxic manifestations in over 20 cases and asserts that mapharsen is much less toxic in debilitated patients than neoarsphenamine or even quinine. According to him, the disappearance of the parasites from the blood is almost immediate. He found that if a chill is to be expected in less than 24 hours, it usually occurs in spite of the injection, but that if it is not due for more than 24 hours, or if the injection is given at the height of a chill, no subsequent chill will occur.

Investigations

On the male continued treatment service of the Utica State Hospital, tryparsamide and bismuth in conjunction with malaria were being used in the treatment of central nervous system syphilis. The malaria was being terminated with quinine, according to the accepted rule, in doses of grs. 15, t. i. d., for three days, and grs. 10, t. i. d., until negative blood smears were obtained on three consecutive days. The administration of quinine was frequently difficult because of toxic manifestations and the uncooperativeness of some patients. Also, many cases did not show the necessary consecutive negative smears for many days. In one recent case, 15 consecutive daily smears were examined before three consecutive negative ones were found. On another, nine were necessary before the patient could be released from isolation. Since the procedure was for 10 days of quinine therapy before starting to take smears, this meant that from the final malarial paroxysm to the release from isolation required 25 days in the first case and 19 in the second.

Therefore it was decided to try the use of an arsenical to accomplish the following purposes: (1.) To remove the parasites from the blood more rapidly and thus shorten the period of isolation necessary after the last paroxysm. (2.) To decrease the toxic manifestations of the treatment. (3.) To increase the ease of administration of the drug used. (4.) To combine antimalarial and antisyphilitic treatment in one drug.

Mapharsen was selected because of Goldman's favorable report and because it was already available and in use for the treatment of systemic syphilis. Doses of 0.06 gms. were used intravenously to terminate the malaria in each case. Weekly doses were then given for 10 weeks to insure the permanent eradication of the parasites from the blood—following Goldman's recommendations.

To date nine cases have been treated by these means with results shown in Table 1.

This compares very favorably with the quinine treatment which had previously required at least 13 days in every case, i. e., 10 of treatment with quinine and three for the smears to be obtained.

TABLE 1

Patient	Days*	Toxic Manifestation
C. S.	3	None
G. C.	3	"
O. I.	10	"
W. P.	12	"
T. N.	9	"
M. B.	4	"
W. S.	5	"
F. T.	4	"
J. M.	4	"
	Average 6 days	

*Number from last paroxysm to release from isolation.

To further illustrate this, a comparison was made with four cases (Table 2) which were interspersed with these nine cases and terminated with quinine because of a known sensitivity to arsenicals.

TABLE 2

Patient	Smears*	Days†	Toxic manifestations
B. T.	3	13	None
P. T.	3	13	Severe tinnitus aurum
P. A	3	13	Severe reduction in appetite
V. Z.	13	22	Pronounced tinnitus aurum
	Average 15 days		

*Number taken before three consecutive negatives were obtained.
†Number from last paroxysm to release from isolation.

Seven of the nine cases terminated by mapharsen showed no recurrent paroxysms at any time during the continued treatment with mapharsen. In two there was a recurrence of fever. One showed a sudden elevation to 105 degrees, 18 days after release from isolation and after four weekly injections of mapharsen. The temperature returned to normal in eight hours with two further spiking elevations of approximately 102 degrees on each of the two following days. The temperature then remained within normal limits; no specific treatment was used during this entire experience. Six blood smears, some taken at the height of the tempera-

ture elevation, were all reported as negative for malarial parasites. The explanation of these phenomena was obscure until at a later check, malarial parasites were found in blood smears two months after this occurrence. It, therefore, seems best to interpret the phenomena as a recurrence of the malaria, despite the use of mapharsen.

The second case of recurrent fever developed a typical malarial paroxym 24 days after the last previous paroxysm, during which time the man had received four doses of mapharsen. This paroxysm was accompanied by demonstrable malarial parasites in the blood stream. To insure proper eradication of the malaria, quinine was then administered in conjunction with mapharsen; and after eight days, the usual three daily consecutive negative smears were obtained.

With individuals known to be sensitive to arsenicals eliminated beforehand, there were encountered no reactions to the mapharsen in any of the nine cases. This compares very favorably with the many toxic reactions, sometimes very distressing, frequently seen with quinine.

The administration of mapharsen is merely that of an intravenous injection and is not dependent at all upon the cooperation or comfort of the patient. It is sometimes almost, if not completely impossible to give quinine orally. Quinine intramuscularly is ordinarily contraindicated and intravenously it may result in severe toxic reactions. Quinine derivatives are also not without toxic properties. The use of mapharsen intravenously also obviates the questionable absorption of quinine in certain individuals.

Mapharsen is now a recognized drug in the treatment of syphilis and is being used for the specific treatment of central nervous system syphilis in some clinics. It thus combines antimalarial properties with an antispirochetal effect.

Young and McLendon[9] report that in dealing with quartan malaria used therapeutically, mapharsen produced remissions of malarial symptoms but did not eradicate the parasites from the blood. Subsequent courses of tryparsamide, those writers found, also failed to effect the removal of the parasites from the blood. Young and McLendon proved the viability of these parasites by sub-inoculations to produce typical malaria. In the present writ-

ers' series, benign tertian malaria was the type under consideration, but to check this point of whether parasites remained after the remission of symptoms, repeated blood smears were examined in three patients who were available for such study. In two, a five months period had elapsed since termination of the malaria; and in the third, two and one-half months. This last patient showed the presence of malarial parasites, and the details of his experiences have been previously described. The other two were negative for parasites. It seems, therefore, that mapharsen brings about remission of symptoms in benign tertian malaria and in most cases eliminates the malarial parasites from the blood. However, in some patients (two of the nine) symptomatic recovery, rather than eradication, occurred.

Conclusion

1. Experience with quinine in the treatment of therapeutically induced malaria of the benign tertian type is at times unsatisfactory, is time-consuming in respect to patient-days in isolation, and is frequently quite distressing to the patient.

2. The possible rôle of arsenical preparations in the general treatment of malaria is not sufficiently emphasized at present.

3. In seven out of nine patients, mapharsen was used successfully to terminate benign tertian therapeutic malaria. In two patients, recurrences of the malaria were encountered despite administration of repeated doses of mapharsen.

4. Mapharsen is superior to quinine in terminating therapeutic malaria of the benign tertian type because: (a) It speeds the elimination of the parasites from the blood in most cases; (b) it is accompanied by fewer toxic manifestations if arsenic-sensitive patients are eliminated first; (c) it is easier and more surely administered; (d) it combines antisyphilitic with antimalarial properties.

5. Mapharsen apparently eradicates the benign tertian parasite from the blood in most patients, as well as bringing about a remission of symptoms.

6. In some patients, mapharsen does not eradicate parasites from the blood and recurrences of malaria do occur. Therefore, it is recommended that 10 weekly injections of mapharsen be used for the termination, that the patients remain under close observation

for one month with occasional blood smear examination, and that when recurrences occur or parasites remain quinine be used in conjunction with mapharsen.

Utica State Hospital
Utica, N. Y.

REFERENCES

1. Stitt, E. R.: The Diagnostics and Treatment of Tropical Disease, 2nd Edition, p. 48, P. Blakiston's Son & Co., Philadelphia, 1917.
2. Cecil, R. L.: Textbook of Medicine, 3rd Edition, W. B. Saunders Co., Philadelphia, 1933.
3. McCrae, T.: Osler's The Principles and Practice of Medicine, 11th Edition, D. Appleton & Co., New York, 1930.
4. Bing and Haymaker: Textbook of Nervous Diseases, translated from the 5th German edition, The C. V. Mosby Co., St. Louis, 1939.
5. Moore, J. E.: The Modern Treatment of Syphilis, p. 356, Chas. C. Thomas, Baltimore, 1933.
6. Henderson and Gillespie: A Textbook of Psychiatry, 4th Edition, p. 325, Oxford University Press, New York, 1936.
7. Beckman, Harry: Treatment in General Practice, 2nd Edition, pp. 99-102, W. B. Saunders Co., Philadelphia, 1934.
8. Goldman, Douglas: The use of mapharsen in the treatment of malaria. Am. J. Med. Sci., 196, p. 502, October, 1938.
9. Young, M. D., and McLondon, S. B.: Treatment of induced malaria in negro paretics with mapharsen and tryparsamide. Public Health Reports, 54, p. 1509, August 18, 1939, as abstracted in J. A. M. A. 113, p. 1601, October 21, 1939.

OPTIC NYSTAGMUS IN SCHIZOPHRENIA

BY MAX A. SHERMAN, M. D.

Introduction

Recently Angyal and Blackman[1] demonstrated that, following caloric vestibular stimulation, the total number and frequency of nystagmus beats is reduced in schizophrenics as compared with normal subjects. It seemed worth while, therefore, to investigate optokinetic nystagmus in schizophrenia, as this function depends on nervous pathways differing from those utilized in the production of vestibular nystagmus.

Optokinetic nystagmus is well adapted for this purpose. The phenomenon is "a conjugate response of the eyes to a succession of moving stimuli which is composed of an alteration of slow and quick phases in opposite direction, when each slow phase consists of the pursuit or fixation of a moving object and each quick phase represents the fixation of a new object of pursuit within the moving field."[2]

Anatomically, the pathways subserving optokinetically induced nystagmus and vestibular nystagmus are identical from the innervation of the extra-ocular muscles to the conjugate ocular center in the pons. There are divergent opinions concerning the nature of the connections of the vestibular and optokinetic pathways above this center. According to the prevailing view they are quite independent.[3, 4] A visuomotor center adjacent to the visual cortex sends corticofugal or optokinetic fibres through the optical radiation to the mid-brain. In addition there is postulated the activity of a frontal lobe center for gaze which may play a part in regulating optokinetic nystagmus and is conceived to have some connection with the occipital visuomotor cortex.[3] On the other hand, Ohm,[5] Spiegel[6] and Smith[7] stress the rôle of subcortical mechanisms in optokinetic nystagmus, particularly of integrations in the vestibular nuclei for determining the slow and fast components.

The character of the nystagmus produced by vestibular stimulation does not differ essentially from that produced by a visual stimulus of moving objects. Dodge and Fox[8] who studied nystagmus produced by both methods, with the mirror-recording photo-

graphic technique, have shown this to be true for normal subjects.

The literature contains reports describing disturbances of optokinetic nystagmus in schizophrenic subjects. Diefendorf and Dodge[9] studied eye-movements of six schizophrenic patients during the pursuit of a pendulum. They reported a "morbid hesitation" of the patients in falling into line with the swinging pendulum (a broken or step-like pursuit) which was regarded as characteristic.

Couch and Fox[10] in a study of eye-movements in different mental disorders reported gross defects in eight out of 43 schizophrenic patients studied, concluding that they point to a "profound incapacity for ocular adjustment well beyond the domain of inattention. Specifically there is shortening, irregularity and elision of the pursuit phases, or still fixation and slow drifting." It must be noted, however, that these patients were either resistive, mute, failed to answer to questions, or failed to respond to command. One is hardly justified in anticipating reliable data in patients of this type who are obviously unable to provide the minimal degree of cooperation required to elicit the responses. Fox also notes that some "sick" patients showed completely normal responses.

Technique

In the present investigation—at Worcester State Hospital, Worcester, Mass.—an ordinary kymograph was used, with a drum six inches in diameter. A paper cylinder, marked with alternate black and white vertical bars, each bar one-half inch wide, was mounted on the drum. An adjustable chin rest enabled the subjects' eyes to maintain a horizontal line of vision with respect to the center of the moving stripes and at a distance of 15 inches from them. A large screen in front of the drum provided a gray background and contained a window limiting the visual field to the width of two bars (one inch). It was possible to maintain the angular velocity of the drum in the range of 33-40° per second (9-11 seconds per revolution). The exact velocity of the drum was ascertained for each reading by recording the time for a complete revolution with a stop watch.

During the preliminary investigation it was found that, due to difficulties with cooperation, the shortest possible test period would

yield the most reliable results. Fifteen nystagmic beats were counted, with the time clocked with a stop watch—in a few patients it was found advisable to reduce the number of beats counted to 10. Under these conditions, gaps in optokinetic nystagmus due to lack of fixation on the surface of the moving drum were eliminated.

The examiner sat to the side of the table facing the patient and close enough to have a good view of the patient's eyes. The lighting was artificial and directly above the center of the examiner's table.

Twenty-eight male schizophrenic patients in a chronically deteriorated state and 15 normal males, all of them attendants or nurses, were used as subjects. The only reason for rejecting either normal persons or patients as subjects in this experiment was the presence of strabismus or myopia. Two patients were not used because their inability to cooperate and fix attention on the moving drum made it impossible to obtain sufficient readings. Patients who would not or could not look at the revolving drum were not used. The gross defects in optokinetic nystagmus obtained by Couch and Fox[10] appear to have occurred in this type of patient.

The drum was in motion while the following instructions were issued to the subject: "Look at the moving black and white bars, try to see them as clearly as possible, and do not let your eyes rest on the sides of the window." In the case of some patients it was necessary to repeat these instructions several times during the experiment. A number of orienting trials was allowed to each subject; and a series of five or more readings was then made. Although the results as presented here are calculated on the basis of the first five readings of each day, use of the entire data available would not materially alter the conclusions drawn. If during a particular reading, the patient blinked excessively, or if his gaze obviously wandered off the drum, the reading was discarded.

The main source of error was in the tendency of the patients to blink excessively or to gaze away from the moving bars. Normal subjects were more consistent in their cooperation and continuously maintained their gaze on the moving surface as directed. This cooperation factor would naturally tend to lower the ratios obtained

in patients as compared with normal control groups. The reliability of the count seemed reasonably above question, since the stimulus used is very impelling, and in most instances the amplitude of the nystagmic beats is such as to be easily observed.

Although the amplitude and regularity of the nystagmus varied from subject to subject, there did not appear to be any characteristic differences between normals and schizophrenics which would tend to an error in any specific direction. The finer characteristics of nystagmus can be studied, however, only by one of the objective recording techniques.[11]

Results

The number of nystagmic beats per second was divided by the number of bars passing by the window in the screen, providing a ratio between the number of nystagmic beats and the number of passing bars. Ideally, the ratio would be one nystagmic cycle for each passing bar. As may be seen from the table of ratios, these are usually greater than unity in both patients and normals. The greater number of nystagmic beats relative to the number of bars is attributable to the refixations within the pursuit phase.[12]

No significant differences were demonstrated in successive readings on the same subject on the same day or on different days, a fact permitting the comparison of the two groups on the basis of

Table 1. Grand Means and Standard Deviations by Days of All Subjects and All Readings in Each Group

	15 normal subjects	28 schizophrenic subjects
First day:		
Mean	1.35	1.20
Standard deviation	.26	.27
Second day:		
Mean	1.34	1.25
Standard deviation	.29	.29

the means for each day. The results are recorded in Table 1. Although the nystagmic frequencies of the schizophrenics are slightly below those of the normals the differences are not statistically significant.

Summary and Conclusions

1. A study of optokinetic nystagmus has been made in 28 randomly-selected patients with chronic schizophrenia and compared with findings in 15 normals.

2. No significant differences in the ratios of nystagmic jerks to the number of passing visual stimuli were demonstrated. It is suggested that differences obtained by previous investigators are attributable chiefly to the inability of some patients to comply with the elementary necessities of the test situation, so that in actuality these patients were not receiving the stimulus. Hence no conclusions can be drawn with regard to their optokinetic functions.

3. In accord with the results of this study, one may conclude that the pathway from the conjugate center of ocular movement in the midbrain to the extra-ocular muscles is not primarily related to the diminished nystagmus reported following vestibular stimulation.

Research Service
Worcester State Hospital
Worcester, Mass.

REFERENCES

1. Angyal, A., and Blackman, N.: Vestibular reactivity in schizophrenia. Arch. Neurol. and Psychiat., 44:611-620, 1940.
2. Fox, J. C., and Dodge, R.: Optic nystagmus. II. Variations in nystagmographic records of eye movements. Arch. Neurol. and Psychiat., 22:55-74, 1929.
3. Fox, J. C.: Disorders of optic nystagmus due to cerebral tumors. Arch. Neurol. and Psychiat., 28:1007-1029, 1932.
4. Kestenbaum, Alfred: Personal communication.
5. Ohm, J.: Zur Augenzitternkunde. 24 Mitteilung 2. Teil (Kortischer Schluss). Graef. Arch. f. Opthal., 126:372-408, 1931.
6. Spiegel, E.: Bemerkungen zur Arbeit von Prof. J. Ohm: "Über die Beziehungen zwischen willkürlichen, optischen und vestibularen Augenbewegungen." Ztschr. für Hals-Nasen-und Ohren-Heilk., 32:471-472, 1933.
7. Smith, K. V., Kappauf, W. E., and Bojar, S.: The functions of the visual cortex in optic nystagmus at different velocities of movement in the visual field. J. Gen. Psychol., 22:341-357, 1940.
8. Dodge, R., and Fox, J. C.: Optic nystagmus. Technical introduction with observations in a case with central scotoma in the right eye and external rectus palsy in the left eye. Arch. Neurol. and Psychiat., 20:812-823, 1928.
9. Diefendorf, A. R., and Dodge, R.: An experimental study of the ocular reactions of the insane. Brain, 31:451-489, 1908.
10. Couch, F. H., and Fox, J. C.: Photographic study of ocular movements in mental disease. Arch. Neurol. and Psychiat., 31:556-578, 1934.
11. Wendt, G. R.: Methods of recording action. Arch. Psychol., 32:5-81, 1938.
12. Fox, J. C., and Dodge, R.: Optic nystagmus. II. Variations in nystagmographic records of eye movement. Arch. Neurol. and Psychiat., 22:55-74, 1929.

PERFORMANCES OF ADULT PATIENTS ON THE BELLEVUE INTELLIGENCE SCALES AND THE REVISED STANFORD-BINET

BY ARTHUR L. BENTON, Ph.D.,* ARTHUR WEIDER, M. A.,† AND JEAN BLAUVELT, M. A.*

The purpose of this investigation was to make a comparative study of the performance of adult patients on the Bellevue Intelligence Scales[1] and the Revised Stanford-Binet, Form L.[2] It was felt that an examination of the relationships between the performances of the same patients on both tests might lead to a better understanding of the tests' function in clinical psychometric practice. Specifically, the degree of relationship between scores of the same patients on both tests and the character of the distributions of scores on the tests have been studied.

Both tests were given to 60 patients, who ranged in age from 16 years to 59, with 31 the median age for the group, and the mean age 35. Thirty-nine subjects were patients at the New York Hospital—Westchester Division, and the remaining 21 were patients at Middletown State Hospital. There were 41 women and 19 men in the group. With respect to diagnostic classification, the following were the important categories: manic-depressive psychosis (19 cases), dementia præcox (16 cases), involutional melancholia (10 cases), psychoneurosis (five cases) and psychosis with mental deficiency (three cases). Only patients who were cooperative and upon whom reliable test scores could be obtained were selected for examination. The tests were given in two sessions, with intervals between sessions ranging from less than one day (tests given morning and afternoon of the same day) to 14 days. In the majority of cases, the interval between tests was one day or less.

Relationship Between Scores. The coefficient of correlation between score on the Stanford-Binet and score on the Bellevue Full Scale was found to be $+.93 \pm .01$. The coefficient of correlation be-

*New York Hospital—Westchester Division, White Plains, New York. Since this paper was written, Dr. Benton has been called to active duty as a lieutenant (jg) in the naval reserve. He is instructor in the School of Aviation Medicine at the U. S. Naval Air Station, Pensacola, Florida, and is psychologist in the medical department.

†Middletown State Hospital, Middletown, New York.

tween score on the Stanford-Binet and score on the Bellevue Verbal Scale was found to be virtually as high ($r=+.92\pm.01$). The coefficient of correlation between score on the Stanford-Binet and score on the Bellevue Performance Scale was found to be $+.73\pm.04$. The coefficients of correlation between score on the Stanford-Binet and score on the Bellevue Full Scale and on the Bellevue Verbal Scale are extremely high, perhaps somewhat higher than one might have expected.* Wechsler[1] (P. 131) found a correlation coefficient of $+.82\pm.03$ between score on the Bellevue Full Scale and score on the Stanford-Binet in a group of 14 to 16-year-old children. In either case, whether one tends to consider the "true" coefficient to be in the "80's" or in the low "90's," it is evident that a high degree of relationship exists between the tests and that they are differentiating these individuals in a very similar way.

Mean Scores and Distributions of Scores. In Table 1 are given the mean I.Q. scores, the standard deviations and ranges of scores of the group for the Stanford-Binet, the Bellevue Full Scale, the Bellevue Verbal Scale and the Bellevue Performance Scale.* It will be noted that while the mean scores are similar, the distributions of scores differ significantly. The standard deviation of the

TABLE 1. BINET AND BELLEVUE SCORES

Test	Mean I.Q.	Standard deviation	Range of I.Q.'s
Stanford-Binet	105	27.7	108 (41-149)
Full Bellevue Scale	100	18.5	73 (59-132)
Bellevue Verbal Scale	105	20.2	81 (55-136)
Bellevue Performance Scale	99	18.4	75 (47-122)

scores on the Binet is considerably greater than that on the Bellevue Full Scale. Likewise, the range of scores on the Binet is considerably wider than that on the Bellevue test. Because of this difference in degree of dispersion of scores, wide divergences in score on the two tests are to be found at both extremes of the distribu-

*The standard deviation of the full Bellevue Scale I. Q.'s is considerably greater than those cited by Wechsler (Ref. 1) (P. 124) for his standardization groups, which range from 13.2 to 16.9 for all age groups and from 14.6 to 15.2 for the 14 to 16-year age groups. The wider degree of dispersion of scores in the present group as compared with Wechsler's groups has the effect of enhancing somewhat the size of the correlation coefficient between the Binet and the Bellevue and is the probable cause of the difference in size of correlation coefficient found by Wechsler and the present writers.

tion of scores. In Table 2, are given the Binet and Bellevue I.Q. scores of the patients who made the five highest Binet scores, the five lowest Binet scores, the five highest Bellevue scores and the five lowest Bellevue scores. It will be noted that for the patients with low I.Q.'s the Bellevue I.Q. is consistently higher than the

TABLE 2. COMPARATIVE TEST SCORES OF PATIENTS MAKING MAXIMAL AND MINIMAL BINET AND BELLEVUE I.Q. SCORES

HIGHEST BINET I.Q.'S			LOWEST BINET I.Q.'S		
Patient	Binet I.Q.	Bellevue I.Q.	Patient	Binet I.Q.	Bellevue I.Q.
A	149	132	G	41	59
B	146	132	H	50	68
C	145	121	I	53	75
D	143	124	J	57	80
E	142	132	K	58	67
F	142	116			

HIGHEST BELLEVUE I.Q.'S			LOWEST BELLEVUE I.Q.'S		
Patient	Binet I.Q.	Bellevue I.Q.	Patient	Binet I.Q.	Bellevue I.Q.
A	149	132	G	41	59
B	146	132	M	62	64
E	142	132	K	58	67
L	141	127	H	50	68
D	143	124	I	53	75

Binet I.Q. and that for the patients with high I.Q.'s the Bellevue I.Q. is consistently lower than the Binet I.Q. It should be emphasized that these divergences in I. Q. score do not indicate a real difference in intellectual capacity, as measured by the two tests, but are solely a function of the difference in degree of dispersion of the scores on the two tests. To state the point in another way, it may be said that widely differing Binet and Bellevue scores at either extreme of their distributions of scores may have the same positional significance, i. e., indicate the same degree of deviation from their respective averages.*

Since this may be the case, it is essential that observed differences in I.Q. ratings of patients by the Binet and Bellevue tests should not be interpreted to mean necessarily that the two tests are

*For a full discussion of the meaning of mental test scores see Peatman (Ref. 3).

not in agreement. It may simply be that scores on the two tests are not comparable because of the differing degree of dispersion of test scores mentioned above. One way of making these I.Q. scores comparable would be by converting them into standard scores or percentile ranks.* Another means of attacking the problem would be to set up a table of "equivalent scores" for the Binet and Bellevue tests, based on the regression equations derived from the correlation between scores on the two tests. Such a table of "equivalent scores," based on the regression equation for the prediction of the Bellevue I.Q. score from the Binet I.Q. score, is presented in Table 3.†

TABLE 3. EQUIVALENT BINET AND BELLEVUE FULL SCALE I.Q. SCORES

P. E.=4.6 I.Q. points

(est. Bellevue I.Q.)

Binet I.Q.	Bellevue I.Q.	Binet I.Q.	Bellevue I.Q.	Binet I.Q.	Bellevue I.Q.
40	59	78	83	116	107
42	60	80	84	118	108
44	62	82	86	120	110
46	63	84	87	122	111
48	64	86	88	124	112
50	66	88	89	126	113
52	67	90	91	128	115
54	68	92	92	130	116
56	69	94	93	132	117
58	71	96	94	134	118
60	72	98	95	136	120
62	73	100	97	138	121
64	74	102	98	140	122
66	76	104	100	142	123
68	77	106	101	144	125
70	78	108	102	146	126
72	79	110	103	148	127
74	81	112	105	150	129
76	82	114	106	...	...

*See Garrett (Ref. 4). (Pp. 177-185.)

†The regression equation for predicting the Bellevue I. Q. score from the Binet I. Q. score is: Y=.63X+34, where Y is the Bellevue I. Q. score and X is the Binet I. Q. score.

The table should be of value to clinical workers for the correct comparative interpretation of Binet and Bellevue I.Q. scores of adult psychiatric patients.

New York Hospital—Westchester Division
White Plains, N. Y.
Middletown State Homeopathic Hospital
Middletown, N. Y.

REFERENCES

1. Wechsler, D.: Measurement of Adult Intelligence. Baltimore: Williams and Wilkins, 1939.
2. Terman, L. M. and Merrill, M. A.: Measuring Intelligence. New York: Houghton Mifflin, 1937.
3. Peatman, J. G.: On the meaning of a test score in psychological measurement. Am. J. Orthopsychiat., 1939, 9, 23-47.
4. Garrett, H. E.: Statistics in Psychology and Education. New York: Longmans, Green, 1939.

THE RORSCHACH METHOD AS A PROGNOSTIC AID IN THE INSULIN SHOCK TREATMENT OF SCHIZOPHRENICS*

BY ZYGMUNT A. PIOTROWSKI, Ph.D.

In a preliminary investigation of schizophrenics treated with insulin shock at the New York State Psychiatric Institute and Hospital, the following conclusion was reached:

"The much improved group had, before treatment, been emotionally much more irritable, labile and impulsive than the unimproved group. However, this relatively greater lability in the much improved group is indicative of a closer emotional contact with the environment rather than of a weaker control over the emotional reactions. For, while the much improved patients had shown a higher degree of emotional lability, they had also disclosed a higher capacity for controlling and stabilizing their emotional reactions because of their relatively better developed inner life and more efficiently functioning intelligence."[1]

A definite psychological difference was found between the recovered or greatly improved patients and the unimproved ones. Before the shock treatment was begun, those who later became much improved or recovered functioned, as a group, on a higher psychological level than the group of those who were unimproved after treatment.

The next step was to study how the knowledge gained from a study of these group differences could profitably be applied to the individual case which, after all, is the main concern of every clinician. Therefore, an attempt was made to determine with some precision the personality level below which insulin treatment was ineffective. The contention that the chance for, and degree of, improvement following treatment vary inversely with the deviation of the patient's personality from the norm, afforded too narrow a basis for Rorschach prognostication when satisfactory results were required in individual cases. It became necessary to add a qualification to this effect: The chances for improvement are greater, the less the schizophrenic's personality has deviated from the norm of healthy adults—but only if the schizophrenic's reactions to the

*Read at the down-State interhospital conference at the New York State Psychiatric Institute and Hospital, April 17, 1941.

environment have been inhibited by a fear of failure, a fear engendered by the feeling that the personality has been unfavorably affected by the disease process. Conversely, when the schizophrenic is making full use of whatever psychological capacities he still possesses, insulin shock therapy does not seem to benefit him.[2] Thus, two psychological conditions for improvement were tentatively formulated: (A) a moderate deviation of personality from the norm of healthy adults, and (B) inhibition of responsiveness to the environment caused by the schizophrenic's fear that the disease process will prevent him from maintaining his former adjustments.

The 'Blind' Prognosis Experiment

It was then decided to submit the tentative conclusions concerning the psychological conditions necessary for improvement to a test by making a series of "blind" prognoses as to the outcome of insulin shock treatment in schizophrenia. Furthermore, there remained the problem of discovering the Rorschach components by means of which the psychological conditions favoring improvement could be recognized most reliably. It was known that, because of other responsibilities of the investigator, the experiment would extend over a long period, and it was hoped that experience gained with the first cases would help to increase the accuracy of predictions for the later cases. The aim was to eliminate the unreliable components gradually and to concentrate on those with the greatest promise of success.

The "blind"' technique was selected because it challenges the validity of the Rorschach method and the skill of the investigator more than other techniques. Rorschach and other data were obtained on 60 schizophrenics, of whom 40 were diagnosed and treated at the New York State Psychiatric Institute and Hospital and 20 at Brooklyn State Hospital. The youngest patient was 16, the oldest 41 years of age; the median age was 23. There were 34 men and 26 women. No selection of patients was made beyond that of choosing as subjects only those schizophrenics who had had no previous pharmacological treatment. All patients studied received hypoglycemic shock treatment later. The study did not include six patients who either refused to cooperate or gave fewer than

five interpretations of the 10 inkblots. The outcome of treatment in each case was estimated by the respective psychiatric staffs, whose opinions served as the basis for classifying the patients as improved and unimproved.

The Rorschach record, obtained from the patient by an assistant, was analyzed "blindly," i. e., without any personal knowledge of the patient (save age and sex), of his history, or of his symptoms, beyond the fact that he was a schizophrenic who was about to undergo insulin therapy. Solely on the basis of the "blind" analysis of the schizophrenic's pretreatment Rorschach record a written statement was offered concerning the probable outcome of the insulin treatment in each case. The predictions pertained to the period of several months after the termination of treatment. The results of this experiment were encouraging. It was found that the predictions as to whether the patient would improve, or not improve, were correct in 88.3 per cent, i. e., in 53 out of the 60 cases. The predictions were correct for 44 out of 48 (92 per cent) of the improved schizophrenics and for nine out of 12 (75 per cent) of the unimproved.

Since clinical estimates were used as criteria for the patients' posttreatment personality changes, one might inquire whether the results would have been the same if the experiment had been carried out in other institutions, with hospital populations not quite the same as those studied, and with psychiatric staffs who might be using somewhat different criteria of improvement. This question can be answered, at least in part, by comparing the results obtained from Brooklyn State Hospital cases with those obtained from the Psychiatric Institute cases, which fell chronologically into two groups. When the first survey of results was made, the outcome of treatment in 21 Psychiatric Institute cases had become known; following this, the prognoses for the 20 Brooklyn cases were written; the final group consisted of the last 19 Psychiatric Institute cases. The predictions were correct in 18 of the first 21 cases (85.7 per cent); in 17 of the 20 Brooklyn cases (85.0 per cent), and in 18 of the last 19 cases (94.7 per cent). The outcome of treatment of the first two groups had been known before predictions for the third were written. The experience gained with the first two raised the accuracy of the predictions for the third. As a matter

of fact, the two patients for whom the most erroneous predictions were made were among the first 10; one patient who recovered had a Rorschach prognosis of "will remain unimproved," while for an unimproved patient a Rorschach prognosis of "much improvement" was given. It is easier to determine whether improvement has, or has not, taken place in a patient than it is to determine to what degree the patient has improved. This is true, not only of the Rorschach method, but also of clinical estimates of improvement. For instance, the 40 Psychiatric Institute cases were divided clinically after treatment into 13 much improved (and recovered), 14 improved, four slightly improved, and nine unimproved patients; whereas the figures for the corresponding classification for the 20 Brooklyn cases were 10, zero, seven and three. For all that, the Rorschach prognoses agreed exactly with clinical estimates of the various degrees of improvement or of lack of improvement in 50 per cent of the Brooklyn cases, and in 55 per cent of the Psychiatric Institute cases.

The degrees of both predicted and actual improvement, as well as the record of lack of improvement were expressed in terms of a graduated four-point scale: "much improved" (including the recovered cases), "improved," "slightly improved" and "unimproved." Thus the disagreements between the Rorschach predictions and the clinical estimates of outcome of treatment could be classified as one, two, and three-point disagreements. For example, a Rorschach prediction of "much improved" for a patient who was considered "unimproved,"' or vice versa, would constitute a three-point disagreement because two intermediary groups, the "slightly improved" and "improved" separate the "unimproved" from the "much improved." Despite the fact that the outcome of treatment was estimated by several physicians (each of whom evaluated only his own patients), there was complete agreement between the Rorschach predictions and the physicians' estimates of treatment in 32 cases, a one-point disagreement in 19 cases, a two-point disagreement in seven cases, and a three-point disagreement in only two cases out of the total of 60.

The Prognostic Signs

It was apparent that the Rorschach method could best serve as a prognostic aid if the procedure of predicting outcome of insulin shock treatment was not too complicated. About 30 Rorschach components were therefore scrutinized in an effort to simplify the procedure. Eventually six components were selected, both for their high predictive value and the ease with which their presence could be determined. These signs are indicators of psychological traits which bespeak a good prognosis. They can be divided into two groups. One group, comprising the signs Vrt, GT, Evd, pertains mainly to certain fundamental intellectual traits; the other group, composed of signs CR, IC, Dmr, pertains chiefly to the patient's sensitivity and responsiveness to environmental changes.

(1) *Vrt* or "variety." The record is credited with this sign if no percept, i. e., the visual image projected into the inkblots, is used more than twice in the interpretation of the blots. If a percept is used three or more times, its first application is usually well justified by the shape of the interpreted ink spot; the percept is usually repeated later when the patient can not think of a new and adequate interpretation and prefers to repeat an old interpretation even though it does not fit its respective blot well. Perseveration in the Rorschach is indicated if the percept perseverated with is misapplied at least once, i. e., if it is projected into an inkblot the form of which does not correspond to it. Vrt has been selected to indicate the opposite of the sign Rpt.[3] Vrt is credited when the patient has not projected the same percept into more than two blots. Frequent repetition of a sexual image, however, is not considered to be intellectual perseveration and is compatible with Vrt as defined here. The reason for this exception is empirical; a number of the much improved and recovered patients perseverated with the same sexual image, vagina or penis, while none of the writer's unimproved patients perseverated with these sexual percepts.

The presence of the sign Vrt is equivalent to the absence of the sign Rpt, which is not infrequent in Rorschach records of patients with cerebral organic lesions.[3] A very large proportion of schizophrenics who failed to improve after insulin shock therapy also

manifested intellectual stereotypy, or Rpt, in their Rorschach records. On the other hand, few of the improved schizophrenics displayed the Rpt sign.

(2) *GT* or "generic term." The record is credited with this sign if it contains proof that the patient has been aware of the difference between generic and specific terms, recognizing the existence of logical hierarchy, and if he has used these terms in a logically correct manner at least once during the Rorschach examination. Correct logical and verbal hierarchy is not identical with visual acuity of form. The patient is credited with GT if he has subordinated at least one specific term to its logically proper generic term regardless of whether the percept to which the terms refer has been acutely or vaguely projected into the ink spots. If a generic term alone is mentioned by the patient, GT should not be credited without ascertaining that the patient had the distinction between generic and specific terms in mind when he used the term. Sometimes expressions such as "some type of bird" or "animal form" are used when the patient is hunting for the name of a specific animal he has in mind, being unaware of the logical relationship between the genus and the species.

Examples: Might be a butterfly or another insect (blot V). A door down here, of a church or a house, an entrance anyway (VII, bottom center). Animal-like thing, rabbit or dog (II, black). Two animals, perhaps a raccoon (VIII, sides). Of course you could not attach any living thing it is, except some sort of . . . it looks like it has a snake's head but a butterfly's body (VI, top part). It could be a butterfly, an insect (I). Well, would the large fish be a whale or is it a shark? (X, pink). A sort of combination of a rabbit and a bat with horns; some characteristics both of an animal and fowl (V). Looks something like a map; looks like the United States (I). Insect with wings, a butterfly (I).

(3) *Evd* or "evidence." The record is credited with this sign if it contains proof that the patient has been aware of the problem of fit, of the question of to what extent the percept corresponds to the physical aspects of the given blot, and if it contains proof that the patient weighed the evidence in support of the adequacy of at least one of his blot interpretations. Evd implies that the patient has been conscious of the distinction between the percept and its respective blot and that he has been aware of the relationship of

correspondence which should exist between them. The sign Evd indicates a concern for evidence and does not necessarily mean that the evidence is satisfactory. Even when the percepts do not fit their respective inkblots adequately, Evd may be credited provided that the patient has demonstrated that he attempted to adjust at least one of his percepts to the objective form (rarely color) of the splotches. Few schizophrenic patients discuss clearly and directly the matter of correspondence between their percepts and the ink spots. The sign is more frequently recognized indirectly by the logically correct use of the disjunctions: "either . . . or," "neither . . . nor," "nor . . . but" and "although." These disjunctions, denoting as they do possibility of choice between several alternatives, are considered to be satisfactory indications of Evd.

Examples: Butterfly, if wings were a little better; a butterfly with a nervous breakdown (VII, bottom detail). Of course it is ink but it gives the appearance of cat; that's the closest I can get to it (I). Slight indication of a butterfly also (VI). From different angles you see different things, the chief characteristic of which is "fowl" (V). This would look like an anteater as much as I can see, or may be a polar bear or a definitely sitting individual, maybe (II, black). This part could be most anything; reminds me of the pelvic bones (III). Could it be other than a mosquito or anything like that (VI)? The way the shadows are, and everything like it might pertain to anatomy or something like that (IV). Couldn't say exactly what it is; could be a part of the chick (III, right black). Possibly lobsters, maybe crabs (X, side blue). It looks like a man, has the figure of a man anyway (III, left black). A dog, or wolf or fox (IV). Looks like a butterfly or a bat; more likely a bat (V). Looks like a police dog in the middle; looks like Christ on second thought (I, middle). Either an insect or a small fur bearing animal, probably an insect (VI).

(4) *CR* or "color response." The record is credited with this sign if it contains at least one interpretation of a chromatically colored blot or of any of its parts, an interpretation which has either a concrete or an emotional connotation, and in which the color has been a contributing determinant. Some responses may apply to a combination of several colored blotches, such as "flower bed," "anatomical slides" or "fairy tale scenes." Any form-color, color-form or pure color interpretation is a CR. Excluded from the category of CR, which includes only meaningful color interpretations, are color denominations.[3] A response is classified as

color denomination if it contains merely the name of the color in description of a spot and if this is considered a satisfactory response by the patient, calling for no additional explanations. The patient must clearly show in his words and by his general behavior that by naming the color he has settled the matter to his satisfaction. Color denominations differ in their psychological and prognostic significance from other types of color reaction. Color denominations are an unfavorable sign in adults, while meaningful color interpretations are a good sign. The chance for great improvement or recovery is extremely small in cases without a CR,[4] but many schizophrenics with slight or moderate degrees of improvement give pretreatment Rorschach records without that sign.

Examples: The back looks like iced tea, the color of it (orange in blot VIII). Reminds me of a person with blood-shot eyes with a crack on top of the skull (top red in reversed II). Some yellow ectoplasm (X). Looks like a frankfurter roll (pink in IX). Is this heaven or earth? (black-red contrast in II). An optimistic outlook on the future! Hope . . . Faith . . . The variegated color (X). What's the point in the red ink? Well, blood, isn't it? (III). Sacred heart (middle red of II). Looks like a smear of big magnified fingerprints and blood (III). A large flower (whole IX). The red thing in the middle looks as though the two were burned in the chest and this meat fell off (III).

(5) *IC* or "indirect color approach." This may be defined in a variety of ways. It is suggested to take the following types of reaction as indicators of IC: (a) Expression of an emotional attitude of like, dislike or doubt in reference to the colors, regardless of whether the colors are interpreted, described or merely indicated by word or gesture. (b) Statement that the colors do not have any meaning, that they are "just colors." (c) Definite evasion of colors, e. g., conspicuous evasion; circumstantial opening remarks or exclamations to inkblot II or to any of the last three, especially VIII; a longer delay between the first glance at the inkblot and the giving of the first meaningful interpretation to it in the case of blots II or VIII than in that of the other blots. (d) The incidental mention of a color's name. This should not be confused with color denominations or Cn[3] which are not included within the IC. Whether a chromatic color blotch is interpreted or merely de-

scribed, the mention of the color's name does not add anything new to the patient's interpretations, is superfluous logically and, therefore, considered an indirect approach to color.

The IC denotes a reaction similar to, although not identical with, Rorschach's color shock.[5] Any manifestations of color shock would be included among the IC responses, but the latter include more than the usual color shock reactions. Naming of, or evasive reaction to, the achromatic colors, black, white, gray, does not belong to the IC class.

It may happen that the same interpretation of a patient may be scored both as CR and IC. E. g., "this red looks like blood;" this is a CR because the red is meaningfully interpreted as blood and it is an IC because the name of the color is needlessly mentioned, adding nothing to the logical content of the interpretation.

Examples of IC: Two yellow spots that look like dogs facing each other between the two red blots (X). Looks like two blue lobsters (X). This red thing on the top looks like a scarf whirling around (II). These are pretty colors (VIII). Might be the colors of the rainbow for all I know (IX). Red and black, that's all; it's hopeless, I can't see anything (II). Just a couple of colors, you know (II). They are just a lot of blotches and colors; there is nothing definite here, they do not have any significance for me (X). Oh, golly! See, all those colors suggest a lot of conglomeration or some activity (IX). I've never seen this before (II). Couldn t say what it is (loud laughter); can't imagine it as anything (II) I refuse to imagine something out of nothing; put down, God damn nonsense in my estimation (red of II).

(6) *Dmr* or "demurring." The record is credited with the sign Dmr if it indicates that the patient has held back one or more of his interpretations in order not to commit himself to interpretations of whose adequacy he is not certain. Dmr is assigned if there is no doubt that the patient during the Rorschach examination has been deliberately careful, has been consciously putting off the communicating of some of his ideas without letting them slip off his mind because he thought exception might be taken to what he was about to say or if he has irresolutely objected to the giving of definite inkblot interpretations. Dmr is not scored when the patient's demurring is only apparent, that is, when there is no conscious caution and fear of failure. Intellectual difficulties also may be responsible for the hesitation and the dubious responses; and

demurring must not be confused with intellectual impotence or Imp;[3] for Imp stands for the giving of interpretations which are inadequate and are as such recognized by the patient, while Dmr stands for the withholding by the patient of interpretations which may be quite adequate.

Dmr is not credited when the patient demurs only in response to the chromatic colors, for this would be an IC reaction. Dmr reflects a more general attitude, characterized by some uneasiness and fear which make the patient wary, make him prefer reflection to action.

Examples: I really don't know; might be a butterfly (IV). Well, if you don't know . . . if you don't know, what do you do then? (I). When I say similar, I say similar to the eye because it's all different material (I). Might look like a bat or lung or something. Do we have to go through with this test now? (IV). That's as far as I can go. The rest looks like a blue (VII). Well, could you give me an example? (I). I think it looks a little like a skin spread out. They don't all look like something very definite, do they? (VI). Is this the right way to look at it, this way? Well, I would not know. I don't think I would know exactly what it is (II). Is this upside down? Is this how it's supposed to be? This looks like a dragon (IV).

The following rule should be observed: If the Rorschach record of a schizophrenic who has had no shock treatment contains more than two of any of the six signs, an improvement can be expected after termination of the insulin shock treatment.

This prognostic rule is based on a comparison of the incidence of the signs in the improved and in the unimproved groups. Altogether, pretreatment Rorchach records of 104 schizophrenics, among which were those obtained in the preliminary and "blind" prognosis experiments, were available. Included, were all patients who, in the opinion of their physicians, had had a fair trial of the insulin shock therapy, who had completed the treatment as it had been planned for them, and who had had no previous shock treatment of any kind. The median age of the total group was 22 years; about two-thirds of the patients fell into the age range of 20 to 30. The youngest patient was 15 and the oldest 41. The outcome of treatment in each case was estimated by the respective psychiatric staff; and their opinions served as the basis of classification as improved or unimproved.

TABLE 1. THE INCIDENCE OF PRETREATMENT RORSCHACH RECORDS WITH THREE OR MORE AND WITH LESS THAN THREE FAVORABLE PROGNOSTIC SIGNS IN AN IMPROVED AND IN AN UNIMPROVED GROUP OF PATIENTS TREATED WITH INSULIN SHOCK

	3 or more signs	Less than 3 signs	Total
Improved	74	3	77
Unimproved	4	23	27
Total	78	26	104

Table 1 shows that if the rule that at least three signs indicate improvement and less than three signs indicate lack of improvement had been used as a basis for prognosis, the predictions would have been correct in 97 of the 104, or in 93.3 per cent of the cases. This is a gain over the 88.3 per cent of correct predictions obtained in the "blind" prognosis experiment (p. 809). This gain was made possible by limiting the number of signs, by describing them with greater precision, and by laying down a more accurate prognostic rule.

The Rorschach examination should be given in the usual manner, i. e., according to Rorschach's directions, to determine which prognostic signs the patient is capable of manifesting. The subject should receive the cards one at a time, always in the same succession, and asked to tell what they look like. The patient is permitted to take all the time he wants. However, experience has shown that a time limit of five minutes a card would be practical; the material contributed after the first five minutes does not add significantly to the personality picture. The subject should look at the cards from a distance of no more than two feet; this is important when examining patients who are confined to their beds and have a tendency to expectorate. Some schizophrenics interpret the blots but refuse to touch the cards. When the patient is negativistic, it is advisable to lay the card directly in front of him and let a few minutes elapse before asking him what the card could represent. It is most desirable that the record be taken verbatim, which is not such a difficult requirement in examining schizophrenics, who, as a rule, do not give very many interpretations. In the present group of 104 patients, the average number of individual total responses was 24. Ovsiankina's 37 schizophrenics gave 26 responses on the average.[6] The average for Halpern's 17 was also 26.[7]

Beck's[8] 81 schizophrenics gave an average of 33. This group, however, contained a considerable percentage of paranoids. These usually give more interpretations than other schizophrenics, mainly by paying a good deal of attention to small details.

The person taking the Rorschach protocol should be well acquainted with the prognostic signs and their psychological definitions, as well as with the fundamental principles of the Rorschach technique.[5, 9, 10, 11] Otherwise, there is danger of missing reactions which are prognostically important. Observation, not only of the patient's verbal responses, but also of his non-verbal reactions, helps greatly in deciding whether a prognostic sign is or is not present. In fact, sign Dmr can hardly be recognized without a scrutiny of the patient's motor reactions. The patient's comments during the inquiry[11] can be utilized to determine the presence or absence of a sign, if the comments are offered freely. No leading questions should be asked. The signs have prognostic value, only if they are produced spontaneously, not in response to direct questions. Sometimes, gestures or a few words of encouragement are sufficient to prompt the patient to elaborate his interpretations given during the first showing of the blots. At times, a smile is all that is needed to elicit further comments. Non-leading questions and interrogative exclamations of the examiner do not invalidate the Rorschach protocol. While it is permissible to exert some pressure in the hope of obtaining at least one interpretation for every one of the 10 inkblots from the patient, the examination should be as free from coercion, as possible, in accordance with Rorschach's[5] own definite instruction.

Besides the six prognostic signs, there are some other Rorschach components which have prognostic significance. These components, however, occur too rarely to be considered of primary importance. Nevertheless, they qualify the conclusions reached through the application of the six signs. An additional component which augurs well is direct response to the black and dark gray colors of the spots, examples of which are "black butterfly," "black animal skin," dark night," or remarks such as this: "I don't like the black in here." Another component strengthening the chances for improvement is the human movement interpretation. Only about one-third of the present group of schizophrenics gave both human

movement and color interpretations; but of these patients, 94 per cent manifested some degree of improvement. A combination of at least four of the main prognostic signs with a black-color response and a human movement interpretation almost certainly presages improvement.

Other components discourage belief in improvement, or at any rate speak against the duration of improvement. They refer exclusively to primary intellectual deficiencies. Contaminatory interpretations, described by Rorschach and Beck, are among the prognostically unfavorable indicators. Proof that, occasionally, during the Rorschach examination the patient completely lost control over his thought processes is also a contraindication of improvement. If any of these additional unfavorable components are prominent, no improvement of marked degree or long duration should be expected.

Discussion and Conclusions

How is one to account for the rather unexpected success of the Rorschach method as a prognostic aid? The clue seems to be in the microscope-like properties of the method. The Rorschach method functions as a psychological microscope by permitting us to distinguish, with greater precision than appears possible by other means, intellectual regression from emotional regression, i. e., the regression caused by primary intellectual deficiencies, from the regression produced by the schizophrenic's fear that his attempts to adjust to the environment may fail, a fear based on the insight that his personality has changed adversely.

The distinction between intellectual and emotional regression is prognostically very important. The writer's experience has shown that the greater the intellectual regression, the smaller the chances for improvement. On the other hand, the greater the emotional regression, the better the chances for improvement after insulin shock treatment. These statements do not appear to be contradicted by other investigations, although the results of the latter are not formulated in exactly the same terms. Lewis[12] has said: "When the regression is acute and takes the form of a confusion with fluctuating contacts with reality and dips into unreality, the prognosis seems to be better than in the slowly developing types."

In a recently published study of 50 schizophrenics, Cheney and Clow[13] conclude: "The dementia præcox patient who has the best outlook for recovery or improvement following insulin therapy is a male under the age of thirty who has had a comparatively adequate pre-psychotic personality, whose psychosis had an abrupt onset with a definite external precipitating cause. He will have been sick less than a year before the institution of treatment; he will have shown an excited catatonic state without evidence of what we have called deterioration, which is defined as consistent lack of attention to personal habits, bizarre behavior, disconnected thinking, and apathy. Conversely, the patient not likely to benefit from hypoglycemic treatment will be a female over 30 who, with an inadequate personality, insidiously and without definite external precipitating cause, has developed over a period of more than a year before treatment a mixed form of dementia præcox with evidence of deterioration."

If one can describe the schizophrenics with slowly developing psychosis as types in whom intellectual regression is not noticeably complicated by emotional regression, and if one can describe those schizophrenics who show a great variability in behavior and who fluctuate in their contacts with reality as types whose relations with the environment are considerably complicated by emotional factors, then the conclusions concerning prognosis, formulated by Lewis, Cheney and Clow, imply that intellectual regression is an unfavorable sign, while emotional regression is favorable.

Special Rorschach studies of insulin treated schizophrenics have also led to similar conclusions concerning the personality traits which bespeak a good prognosis. Graham,[14] having studied 31 pretreatment Rorschach records, believes that schizophrenics who benefit from insulin treatment do better in detecting logical absurdities and give more responses determined by the *chiaroscuro* aspects of the Rorschach inkblots. These *chiaroscuro* reactions are a classical Rorschach indication of emotional inhibition and are implied by our IC or Dmr signs. Halpern,[7] working with 17 patients who were examined before and after treatment, confirmed the results of the writer's preliminary investigation by stating that patients who improved after treatment had pretreatment personalities functioning on a higher level; she said that the improved

group differed from the unimproved by a greater productivity, wider emotional range and a greater capacity for empathy. The writer's finding that many Rorschach records in the unimproved group resemble records obtained in organic brain diseases[1] was also corroborated by Halpern. These results are welcome, because they support not only the writer's conclusions but also his manner of handling the Rorschach data. When Halpern and her collaborators[15] had treated the data in a different way, they found that the Rorschach had only a low forecasting value.

Three of the Rorschach prognostic signs can be regarded as indication of lack of intellectual regression. Patients who manifest the signs Vrt, GT and Evd have the following assets as compared with the patients who do not manifest those signs: They are capable of producing a greater variety of ideas, they can function on a higher logical level; being aware of the distinction between specific and generic terms, they can compare one idea with another; finally, they show understanding for one essential condition of adequate thinking—the need of verifying their thoughts by empirical evidence. Patients with the CR sign are more sensitive to environmental changes than those without CR; thus, this sign implies a higher level of psychological development. The signs IC and Dmr may be conceived as measures of emotional regression, since they indicate reluctance to give definite emotional and intellectual responses for fear of failure.

The psychological changes contingent upon improvement following insulin shock therapy seem to consist in the reduction or removal of what has been defined as emotional regression. Rorschach records taken after treatment do not show any noticeable improvement in the basic intellectual capacities, but they do show plainly a decrease in emotional irritability and a greater ability to make adequate use of whatever capacities the patient possesses.[2] One might say that successful insulin treatment makes the patient less fearful of contacts with the environment. Cheney and Clow[13] state that patients who benefit by insulin treatment have the same characteristics as those benefiting by other forms of treatment. They express the conviction that "insulin does not have a specific curative effect but that it may bring about changes that accelerate or facilitate improvement in those who have the constitutional capa-

city for such improvement or recovery." Assuming this to be true, it is reasonable to expect that, with some modification, the Rorschach prognostic signs could be profitably used also in other forms of treatment than that by insulin in schizophrenia. The apparent success of the Rorschach prognostic signs encourages the opinion that these signs would be helpful in the selection of patients for insulin shock therapy and probably for other forms of treatment as well.

ACKNOWLEDGMENT

It affords the writer great pleasure to thank Dr. Nolan D. C. Lewis, professor of psychiatry in Columbia University and director of the New York State Psychiatric Institute and Hospital, for his active and helpful interest, without which this investigation could not have been carried out.

Department of Psychiatry
College of Physicians and Surgeons
Columbia University
New York, N. Y.

REFERENCES

1. Piotrowski, Z. A.: The prognostic possibilities of the Rorschach method in insulin treatment. PSYCHIAT. QUART., 12:679-689, 1938.
2. Piotrowski, Z. A.: Rorschach manifestations of improvement in insulin-treated schizophrenics. Psychosom. Med., 1:508-526, 1939.
3. Piotrowski, Z. A.: The Rorschach inkblot method in organic disturbances of the central nervous system. J. Nerv. and Men. Dis., 86:525-537, 1937.
4. Piotrowski, Z. A.: A simple experimental device for the prediction of outcome of insulin treatment in schizophrenia. PSYCHIAT. QUART., 14:267-273, 1940.
5. Rorschach, H.: Psychodiagnostik. H. Huber, 3rd ed., pp. 255, Bern, 1937.
6. Rickers-Ovsiankina, M.: The Rorschach test as applied to normal and schizophrenic subjects. Brit. J. Med. Psychol., 17:227-257, 1938.
7. Halpern, F.: Rorschach interpretation of the personality structure of schizophrenics who benefit from insulin therapy. PSYCHIAT. QUART., 14:826-833, 1940.
8. Beck, S. J.: Personality Structure in Schizophrenia. Nerv. and Men. Disease Mon., New York, pp. ix+88, 1938.
9. Rorschach, H., and Oberholzer, E.: The application of the interpretation of form to psychoanalysis. J. Nerv. and Men. Dis., 60:225-248, 359-379, 1924.
10. Beck, S. J.: Introduction to the Rorschach Method. Am. Orthopsychiat. Asso. Mon., New York, pp. xiii+278, 1937.
11. Klopfer, B.: The technique of the Rorschach performance. Rorschach Res. Exch., 2:1-14, 1937.
12. Lewis, N. D. C.: Research in Dementia Præcox (Past Attainments, Present Trends and Future Possibilities), pp. xi+320, New York, 1936.
13. Cheney, C. O., and Clow, H. E.: Prognostic factors in insulin shock therapy. Am. J. Psychiat., 97:1029-1039, 1941.
14. Graham, V. T.: Psychological studies of hypoglycemia therapy. J. Psychology, 10:327-358, 1940.
15. Wechsler, D., Halpern, F., and Jaros, E.: Psychometric study of insulin-treated schizophrenics. PSYCHIAT. QUART., 14:466-476, 1940.

PRACTICAL PROBLEMS ARISING FROM TUBERCULOSIS SURVEYS IN MENTAL HOSPITALS*

BY O. ARNOLD KILPATRICK, M. D.

This paper discusses the origin and technique of an X-ray survey for pulmonary tuberculosis conducted at the Willard State Hospital since October, 1938, and presents the principal findings and some recommendations. As a basis for comparison, the writer has selected the report of a similar survey of all the Ontario hospitals.

The need for such a survey became evident in two ways. The staff of the Biggs Memorial Hospital—a State Tuberculosis Hospital opened in Ithaca in 1936—noted that an unduly high proportion of admissions came from the region of Willard State Hospital. Seven active cases of pulmonary tuberculosis with respiratory symptoms presented themselves among the State hospital employees within 18 months. Twelve of 81 tuberculosis cases on the register from Seneca County were former or present employees. Tuberculosis is considered compensable. The approximate cost to the State for these employees may amount to $25,000 a year. For the entire State in the next fiscal year, tuberculosis compensation may reach $1,000,000. Within Willard State Hospital, at the same time, physicians were expressing concern at the lateness with which tuberculosis was being diagnosed, a matter constituting a risk to both patients and employees exposed to unrecognized open cases. Active measures on a large scale were called for.

If physical signs and symptoms are waited for, the opportunity to prevent spread and to treat successfully will be missed. This, in effect, was the situation which prevailed before the survey. Various methods that have been used in detecting cases of tuberculosis in large groups were considered. At first the tuberculin test was tried; it was thought that only positive reactors would need to be X-rayed. When 90 per cent, instead of the estimated 40 per cent, gave positive reactions, the number which could be eliminated became insignificant.

*Read before the up-State interhospital conference at Utica State Hospital, Utica, N. Y., April 25, 1941.

The fluoroscope is of value as an eliminative method in expert hands, and is much more efficient than physical diagnosis alone. Among the State hospitals that have used it are Marcy, Brooklyn, Rockland and Middletown. It was decided that its fairly high percentage of error—12 to 15 per cent in minimal cases, according to Fellows of the Metropolitan Life Insurance Company—made it unsuitable for the purpose. Also, it is not applicable to bedridden patients, in whom was found a higher percentage of tuberculosis than in ambulatory groups.

It was felt that X-ray films of the total population of the hospital would limit the error—which is extremely small—to the breaks in technique which must necessarily occur in dealing with mentally ill patients. Films 17″ x 14″ were used. These cost the State approximately 60 cents each. Allowing 10 cents for developing, this item of film cost—always a practical consideration—came to a total of $2,700. The work of the survey was done by the staff of Biggs Memorial Hospital. Willard supplied the films, one technician, and the cooperation of its staff and laboratory. In addition to the cost of films, technicians' time would amount to $300 or $400. Interpretation, in which is included the experts' time, can be estimated at $1,000. The clerical work might involve an expenditure of $500. An approximate figure for the initial survey would be somewhere between $4,000 and $5,000. The actual cost to Willard State Hospital—eked out from Estimate 11—was for materials alone. In comparison to the economic loss sustained through illness of employees, this is insignificant .

A single film is of considerably less value than serial films taken at intervals to demonstrate changes and activity. Most of the patients have now had two or three X-rays, covering a two-year period. As yearly surveys are needed for this and for the detection of new cases, the use of smaller films has been investigated. The army is now using a new 4″ x 5″ film costing six cents. In the opinion of experts, this is adequate, with the reservation that vague lesions of the apices may be obscure. The 35 mm. films have been found inadequate because of lack of adequate magnifying lenses. A comparison of the three types of film at Binghamton State Hospital showed the 4″ x 5″ films were satisfactory but that only 260 of 1,000 of the 35 mm. films could be read.

The definitions of "minimal," "moderately advanced" and "far advanced," as used in this report are those published by the National Tuberculosis Association.[1] An active lesion is one with cavitation, with change in serial X-rays indicative of activity, or with findings of an exudative lesion of apparently recent origin if only a single film is available. Arrested lesions are those appearing older, quite fibrous in character, without change in serial X-rays, and associated with negative sputum. The apparently healed group are those fibro-calcific lesions appearing to have been at least minimal in extent at their origin. These are differentiated from the group called calcification, which includes single nodules of calcium in the lung parenchyma or tracheobronchial lymph nodes.

Of 794 employees surveyed, eight were found to have clinically significant pulmonary tuberculosis not previously diagnosed. In addition, two already had been known to have the disease. Of the eight, six were in the minimal stage, one was moderately advanced and one far advanced. Five of these eight were found to have positive sputum. One with negative sputum showed an unstable lesion on serial X-rays. There were 18 additional employees diagnosed as having a definite reinfection type of pulmonary tuberculosis, with the roentgenological appearances of healed lesions. Seventy-four showed evidences of calcification of the lung parenchyma or of tracheobronchial lymph nodes which were presumed to be of tuberculous origin.

In September, 1940, a one-year followup revealed three new cases of pulmonary tuberculosis. These were diagnosed as radiologically negative in the first survey; and during a period of one year, they had shown sufficient change to be noted as minimal lesions. This demonstrates what has been found elsewhere—that a single screening is not enough, and that annual X-rays will detect cases early enough to make possible prompt and effective treatment.

In the first survey of the patients, 3,407 X-rays were taken of 3,317 subjects. Seventy-six (2.2 per cent) showed active lesions. One hundred and fifty-seven (4.6 per cent) showed lesions that were arrested or apparently so. One hundred and ten (3.2 per cent) had apparently healed lesions. Three hundred and fifty-one

(10.3 per cent) showed calcification of the lung parenchyma or hilar nodes. The remaining 2,623 (79.7 per cent) were negative.

Of the 694 patients showing evidence of tuberculous infection, 11 per cent exhibited lesions that were probably active; 23 per cent were probably inactive; 16 per cent were classified as apparently healed; and 50 per cent showed posttuberculous calcification. When age incidence was checked, it was found that the peak in the active group occurred between the ages of 35 and 44, followed by a gradual decline, then a sharp rise at 75 and over.

At the time of reexamination, there were 72 patients in isolation: 25 had minimal, 26 moderately advanced and 18 far advanced lesions. Two hundred and fifty-two patients were in an observation group: Of these, 27 were moderately advanced, 127 had lesions of minimal extent, and 98 had minimal lesions apparently healed. Of 2,716 patients reexamined, 2,414 were without tuberculosis. During the interval between surveys 16 cases of active pulmonary tuberculosis had developed—14 minimal, one moderately advanced and one far advanced. The annual morbidity rate was thus .66 per cent.

The three buildings in which the greatest incidence was found house a very high percentage of regressed schizophrenics. The survey findings support those of many other investigators, that the highest incidence of tuberculosis is found in the schizophrenic group. Though 67 per cent of the cases occurred in the dementia præcox patients, they constitute only 52 per cent of the hospital population. The majority of these patients were in the hebephrenic and catatonic types. To insure accuracy and uniformity of diagnosis, the writer interviewed personally all patients with doubtful diagnosis, especially those originally classified as manic-depressive psychosis, alcoholic psychosis or psychoneurosis.

The initial findings of the X-ray examination of 13,257 patients in all the Ontario mental hospitals[2] showed 1.3 per cent of active pulmonary tuberculosis, definitely lower than the Willard finding of 2.2 per cent. In general, the further findings, as far as they are statistically comparable, show no marked divergence. The follow-up surveys disclosed 110 new cases—as compared with 476 in the first survey. Ten far-advanced cases had developed within the year—indicating the necessity of reexamination annually. Four of

these have now been made in Ontario, involving 13,000 X-rays each year. In the last three surveys, the new active cases found have numbered 33, 42 and 50—whereas originally, there were 165 active cases disclosed. One fact stands out from these figures: There continues to be a definite percentage of active cases discovered each year by the use of the X-ray which would probably not otherwise be demonstrable. It may be speculated that when contacts are greatly reduced, there remain a certain number liable to endogenous reinfection.

The Ontario findings led Wicks to the following significant conclusions. Though decreasing since 1934, the mortality rate in the province's mental hospitals in 1936 was 14 times greater than that of the province as a whole. Two and five-tenths per cent of 2,908 admissions during 1938 required isolation because of chest X-ray findings. The continued appearance of new cases in subsequent surveys indicated the need for periodic "combings" of the hospital populations. Six-tenths of 1 per cent of 2,542 patients and 0.5 per cent of the employees have required treatment for tuberculosis each year. To control the incidence in staff members, the tuberculin test and serial X-rays are used. A central tuberculosis mental hospital has been recommended to accommodate approximately 5.2 per cent of the patients in Ontario mental hospitals.

After a survey has detected which patients need to be removed from the general hospital population to prevent the spread of tuberculosis, the problem of their proper care and treatment arises. In line with the Ontario solution, the Mental Hygiene Department has, nearing completion, at Central Islip, a tuberculosis unit for patients in the metropolitan area State hospitals who need isolation and treatment for tuberculosis. For the up-State areas, the answer to the problem is less apparent.

The two buildings previously used for the tuberculous at Willard were too small and entirely unsuitable for the purpose, and have been evacuated. So far, it has been necessary to clear two floors of a new infirmary building. One floor has active cases; and the other houses suspicious cases under direct observation. These four wards previously were used for the elderly and infirm men and women; and their loss has been acutely felt. As a permanent arrangement this is not felt to be satisfactory. The ability of the

staff of a mental hospital to care for and treat all forms of active tuberculosis is likely to be limited and inadequate because of lack of specialized training and absence of proper housing and treatment facilities. The situation does not warrant intensive staff training for a relatively few cases. Also, the removal of open cases from the wards is bound to lead to a reduction in the number of cases needing the specialized treatments for open lung lesions.

The establishment of a centrally located tuberculosis unit to care for all active cases in the up-State areas would be, in the writer's opinion, a questionable procedure. Relatives from all over this section of the State—who could ill afford it—would be put to additional expense in their visits; and this would undoubtedly lead to strong protests. The geographical distribution of the up-State hospitals is not well suited to such a plan. To staff a new hospital with experts, as it should be staffed, would be difficult. The care of tuberculosis would have to be combined with skilled psychiatric attention. Classification of patients by any other than psychiatric criteria leads to the grouping together of very dissimilar types of mental problems—which are usually not found on the same wards —making management very troublesome.

Willard has, about 20 miles from its door, a new, modern, well-equipped State hospital for the care of pulmonary tuberculosis. It is staffed by experts who have willingly given their services in the solution of Willard's problem. The best solution of the problem of the tuberculous patients, as far as Willard is concerned, would be the building of a psychiatric wing at Biggs Memorial Hospital to which active cases and those requiring surgical attention could be sent. There they would receive the necessary skilled care, and would benefit by the use of facilities not now available to them. A qualified chest surgeon is available there. The nursing and supervisory care could be in the hands of a psychiatrically-trained personnel. The observation group could be cared for at Willard, with the assistance of an annual survey. With the checking of new admissions, and examinations of new staff members and employees, the situation would then be under permanent control.

Summarizing, the experience of the Willard survey suggests the following measures may be applied in controlling tuberculosis in mental hospitals:

1. All patients and employees, including staff, should have chest X-rays at yearly intervals.

2. Cases showing anything suspicious of activity should be X-rayed every six months, or oftener if indicated.

3. All new patients should have X-rays on admission.

4. All applicants for employment on the staff or as employees should be X-rayed when they make application.

5. Proper public health measures should be rigidly enforced in the segregation and care of any type of case shown to have tuberculous infection.

6. Staff and employees caring for any type of tuberculous disease require careful instruction about the nature of the disease and about the practical measures they should adopt in nursing procedures.

7. All employees should be X-rayed on leaving the service. This may be important in compensation cases.

8. Cases under treatment require proper records. Simple forms used at Willard condensing all information relevant to tuberculosis on one page, including X-ray reports, have been found very useful in controlling treatment and in gathering data pertinent to the whole group under care.

9. There is a definite need for some form of expert care for active tuberculosis cases in State hospitals.

10. Establishment of a central unit for up-State areas should not be undertaken without a careful consideration of all the factors concerned.

11. The provision of added facilities, in tuberculosis sanatoria already existing, for the care of psychiatric cases suffering from active tuberculosis is worthy of consideration.

12. A traveling clinic might be organized to make annual surveys in all State hospitals. It should consist of a qualified specialist in tuberculosis, expert technicians and a portable X-ray unit.

The saving in lives, health and money would thoroughly justify measures to control and prevent tuberculosis in the mental hospitals of New York State.

Willard State Hospital
Willard, N. Y.

REFERENCES

1. Diagnostic Standards and Classification of Tuberculosis: National Tuberculosis Association.
2. Wicks, C. A.: Am. J. Psychiat., 96:6, May, 1940.

BOOK REVIEWS

The Development of the Sciences: Second Series. By Oystein Ore, Frank Schlesinger, Henry Margenau, John Arrend Timm, Chester Ray Longwell, Lorande Loss Woodruff, Walter Richard Miles, John Farquhar Fulton. Edited by L. L. Woodruff. 336 pages. Yale University Press. New Haven. 1941. Price $3.00.

The Yale Chapter of the Gamma Alpha Graduate Scientific Fraternity arranged in 1939 a series of lectures on the history of science to be given by members of the science departments of Yale University. Eight lectures cover the following fields: mathematics, astronomy, physics, chemistry, geology, biology, psychology, medicine and the sciences. Engrossed as the scientist is in contemporary problems, he is often prone to regard the history of his subject as an affair of accepted clichés or unprofitable romancing. This admirable volume, however, succeeds in making of the history of the sciences something far better than a rhetorically embellished account of the curiosities of outmoded opinion about nature and the universe.

The essay on mathematics by Professor Oystein Ore, that on psychology by Professor Walter R. Miles, and that on medicine and the sciences by Professor John F. Fulton make a particularly favorable impression. Professor Ore summarizes the complex history of mathematics, from Babylonian mensuration and business arithmetic to the latest developments of mathematical logic, in an exceptionally lucid and economical English style. The difficult field of academic psychology—really a congeries of some score of psychological systems—is excellently presented by Professor Miles without any apparent sectarian bias. The present glaring lack of synthesis in psychological science may leave the reader, as Professor Miles would probably agree that it should, with the impression that psychology has, to date, perhaps succeeded only in accumulating the bricks and mortar out of which the psychologists of the future will construct a stable edifice. The concluding paragraph of this lecture seems well worth quoting in full:

"Today—as in the evolutionary century, during the Renaissance, in the early Christian era, and in the Greek period—psychology faces the same fundamental problems: the mechanisms and dynamics of human behaviour; external and internal influences; inner integrations in awareness, memory, drive, and readiness for response; and finally the differences in individual structure and function that distinguish one personality from another and are the stuff of moral character and social progress. Successive generations of observers and experimenters have diligently labored to disclose essential

facts. We are in no position to say how much progress has actually been made. Through the measurement technique, psychological facts, laws, and relationships that we designate as scientific are now demonstrable by routine experiments. The large problems of man's mind are still perhaps no more than glimpsed, and intelligent control of his behaviour through conditioned social integration is still far before us."

The vast subject of the history of medicine was not discussed for its own sake. Unfortunately so, it might seem. Professor Fulton, however, has presented the history of medicine as the matrix from which came enormously important contributions to the development of the other sciences. The burden of this essay, namely the thought that it has been, in the past, again and again the physician who made fundamental contributions to pure science, is an interesting one, and it is perhaps Professor Fulton's lecture which could make a more reasonable claim to originality than anything else in the volume. Medicine, it seems, with its practical orientation, a discipline which has rarely been tempted to regard itself merely as a "nosology," or doctrine of disease, has survived, and perhaps in some sense even flourished, in times which were otherwise either impatient of free speculation and experiment or impressed only with the authority of ancient worthies such as Plato and Aristotle.

Scientists are human beings, and the products of their thinking have their existential ground in no disembodied intellect but in an institutionalized form of human behavior, and that, too, a fragile, perhaps even an ephemeral one. A science which essays to believe that it can or ought to insulate itself from the broad pattern of the culture which supports it is living in a fool's paradise. In rare and fortunate epochs scientific curiosity has been tolerated and even encouraged. It is perhaps more usual, though, to find the common run of mankind inclined to show the scientist that he is not the master. The cold fact is that human society has emotional and instinctive interests which frequently conflict with what we please to call scientific objectivity. These interests can, and on occasion will, lead to the ruthless sacrifice of the scientist's preference for objectivity—itself, too, an emotional diathesis of the psyche. As the history of Germany in the twentieth century all too clearly shows, modern popular education offers no panacea for the fatal results of such a situation.

Professor Margenau, in his lecture, may regret, if he chooses, that lack of magnanimity, good taste, or enlightenment which led to the burning of Giordano Bruno, but such regret has little bearing upon the history of the physical speculations of the Middle Ages and the Renaissance. In fact, not a few of Professor Margenau's statements do little credit to his discrimination as a historian or his understanding of the rationale of social be-

havior. A scientist who proposes to ask tolerance for his own work should first understand those instinctive sides of human nature which express themselves in institutionalized religion and institutionalized political behavior. Greek physics is really metaphysics; with the Stoics physics amalgamated in a very questionable way with the theory of morals; and with the mediaeval Christians it tended toward an unstable union with theology. For Professor Margenau this is all very bad; unfortunately it is all very human, and if one does not care to understand it as such, it is perhaps wiser to begin the history of physics with Newton's *Principia*. A few scientists working from quite different points of view, such as Duhem and Carl Jung, have been able to read themselves into documents of ancient physics, astrology, alchemy, mystical theology, and have sensed something of their original meaning as living expressions of man's perennial quest to understand himself and the world in which he lives. Professor Margenau is not among this number. A paragraph which I quote is perhaps a classical example of the way not to write the history of ancient physical speculation:

"The dazzling perfection of the life of Christ stunned his followers into the conviction that his moral teachings were divinely inspired, and St. Paul, in doctrinizing his teachings, succeeded in enlarging this conviction into the belief that natural knowledge, too, must spring from revelation. Greek writings were inaccessible during the first centuries of the Christian era, centuries in which scientific thought groped erratically for supernatural and absolute truth. Later, with the thorough establishment of Paulinism and the rediscovery of Greek philosophy, the stage was set for the elevation of one sage upon the pedestal of scientific infallibility, and the church fathers had no difficulty in selecting one whose philosophy was most closely in harmony with their conception of Paulinistic doctrine. And so it came to pass that Aristotle's physics as well as his philosophy dominated the Middle Ages."

The complex thought currents of the ancient world, particularly the Hellenistic age, are too elusive, the concepts which they employ too highly overdetermined, to allow anyone who approaches them at second hand to hope to generalize intelligently about their meaning. The mind of the Hellenistic age was not our mind and its thoughts were not our thoughts, and those who have not the patience to understand are well advised not to tarry over its theories of nature. The conscientious study of ancient documents is always costly of time and effort. Where a first-hand approach is impossible, one can do no better than recommend the reading of Carl Jung's chapter on alchemy in his "Integration of the Personality." Even though one were not prepared to accept in full Jung's conclusions, one can hardly fail to gain from his discussion a sense of the depth and complexity of the

problems which underlie all interpretations of ancient documents in the fields of physics, astrology, alchemy, magic, mystical theology, and cosmology.

When Professor Margenau is on ground with which he is familiar, he is extremely cogent and always clear. His discussion of wave mechanics and the structure of the atom contrasts very favorably with the usual attempts to popularize these matters. Here he escapes entirely the misleading impression made by so many who write about modern physics, the impression, namely, that Einstein and his brilliant satellites somehow mean to take physics back to mysticism. "Light," says Professor Margenau, "and matter are neither particles nor waves. Their basic nature can be understood only in more abstract yet perfectly definite terms. In retrospect, this conclusion seems almost obvious, for is it not true that light waves and electrons are so small that the question as to their shape can never be answered? . . . As for the electron, to picture it round and of exact diameter would be just as ill advised as to picture it blue." Professor Margenau has shown that a contemporary physicist can, if he will, talk in a way which appears sensible even to a layman.

The other essays in the volume, that on astronomy by Professor Schlesinger, on chemistry by Professor Timm, on geology by Professor Longwell, and on biology by Professor Woodruff, are excellent and do not deserve the summary dismissal which this review must allot to them. The volume as a whole merits the attention of all who are interested in the historical background of modern science.

Psychiatry as a Career. One of a series of pamphlets issued by The Institute for Research devoted to vocational research, 537 S. Dearborn Street, Chicago. Bound pamphlet. 1941. Price $1.00.

This is an attempt to present to the unitiated advantages and disadvantages of psychiatry as a career. It begins with an historical introduction and traces the origins of psychiatric interest and study in western Europe down to the work of von Jauregg and Manfred Sakel. The latter half of the pamphlet is given over to the description of the organization and work of modern psychiatric hospitals and clinics. It was presumably prepared by the editorial board of which John A. Lapp, LL.D., is the editorial director.

The text is couched in terms of moderation and it is a monograph which might properly be placed in the hands of a young man seeking advice as to a career in medicine.

Time and the Mind. Personal Tempo—The Key to Normal and Pathological Mental Conditions. By HARRIET BABCOCK, Ph.D. 304 pages with index. Sci-Art Publishers, Harvard Square, Cambridge, Mass. 1941. Price $3.25.

Prior to the advent of insulin and shock therapies, psychiatry was showing dangerous tendencies. It was no longer entirely popular to denounce patients by reproaching them for various stigmata of degeneration, unfortunate heredity, and constitutional psychopathic inferiority. Vague murmurs began to imply that the retention by society of its feudalist ethical codes might be responsible for some failures of the individual, and that it might be as profitable to investigate the system under which a person had to live, as to assay his hormones. However, the reaction had its way, and mental illness once more became something to be exorcised; became only a fault of the patient, to be removed by the magic of insulin and metrazol.

Dr. Babcock also represents a return to the old time religion, but in a different way. There is a company of the elect, (those who get along well under present circumstances), and a company of the damned, (those who, because of structural faults in themselves, fail of making an adaptation to this best of all possible worlds). To quote from "Time and the Mind," p. 103, "The præcox defect is usually constitutional," i. e., the patient is one of the damned. In speaking of borderland conditions, on the same page, she states, "It is a general mental weakness undoubtedly due to a neurological or somatic condition, . . ." The use of words like "undoubtedly" is hardly permissible in science, and is entirely out of place in discussing the relationships of the psyche and soma, in the present state of our knowledge. If the facts are thus and so, an "undoubtedly" will not make them any more true. If they are not thus and so, an "undoubtedly" is subversive propaganda.

Aside from her gratuitous remarks about the basic nature of mental illness, the author seems to have written a sound report concerning the results of the application of various batteries of tests to various groups of people, and to have reached sufficiently aimiable conclusions as to their parolability, employability, etc., on the basis of these tests. Thus far, the report is useful, stimulating, and practical. However, having found that mental malfunctioning is almost uniformly associated with disturbances of mental tempo, which can be measured, the author seizes on this attribute of maladjustment and makes it the cause of maladjustment. That kind of thinking has led to great mistakes in the past, one example of which was national prohibition.

Dr. Babcock has probably started something. There is a market for the exposition of any method which will attempt to settle psychiatric questions numerically or graphically. It saves thinking and shifts responsibility. Furthermore, research in mental tempo should serve to keep large numbers of students inoffensively occupied. They will not get any wrong ideas from "Time and the Mind." In a book which purports to be "The Key to Normal and Pathological Mental Conditions," there are two pages devoted, half-heartedly, to sex. On one of these, (p. 258), there appear some remarkable statements. "The importance of sex has been well recognized since the dawn of history as is shown by the numerous taboos and customs, which seem to have been an intelligent way of handling the problem. People adjust to different customs because of unconscious acceptance of the rightness of the ideas inculcated by their society. These ideas preclude expectation and lessen mental stimulation to the sex impulse." The reviewer rests.

The 1940 Year Book of Neurology, Psychiatry, and Endocrinology. By Reese, Lewis and Saveringhaus. Year Book Publishers. Chicago. Price $3.00.

The 1940 Year Book of Neurology, Psychiatry, and Endocrinology is a practical and interesting volume of 856 pages, containing an anniversary preface, a table of contents, an index and sections on neurology, psychiatry and endocrinology. These sections are edited by Reese, Lewis, and Saveringhaus respectively. That on neurology includes pertinent information from papers on neurological problems, embracing, among other things, brain tumors, syphilis of the nervous system, muscular disorders, inflammatory diseases of the nervous system, anatomic and physiologic contributions, together with neuropathology.

The section on psychiatry is composed of condensations of the more important psychiatric contributions. In their scope, are such subjects as child psychiatry; general considerations of the schizophrenic psychosis and its treatment, including pharmacological shock therapy and electric convulsion therapy; affective disorders; organic reactions; the toxic psychoses; psychoneuroses and military medicine. Finally, endocrinology is considered from the standpoint of endocrine disorders of the female reproductive system, the pituitary group, and the thymus.

This entire meaty work contains the best of the new thoughts and findings in the fields it covers. The editors append timely remarks to some of the articles to clarify any moot questions which arise—in this way giving the book even greater value. It should be purchased by every physician interested in neuropsychiatric and endocrinologic medicine; and the reviewer heartily recommends it.

A Short History of Psychiatric Achievement. By NOLAN D. C. LEWIS, M. D. 275 pages. Cloth. W. W. Norton and Company, Inc. New York, 1941. Price $3.00.

In the 10 years since they were established, the Salmon Memorial Lectures have brought out a number of important and timely books. The present volume from the pen of Dr. Nolan D. C. Lewis is no exception. The author has rendered a useful service by tracing the history of psychiatric thought and practice through the ages and into modern times and has given indications for further progress and development. The book is a review of psychiatric thought and theories which lie at the basis of modern psychiatry and psychology. The author, who has gained distinction as director of the New York State Psychiatric Institute and Hospital, is primarily a pathologist but is equally learned in clinical psychiatry and now demonstrates new qualities as historian and philosopher. His point of view is mainly that of the philosopher. In this book, he traces the development of psychiatry but sees that there was beneath the surface, even long ago, the stirring of vast movements which have brought psychiatry to the forefront of medical science.

More than any other of the biological disciplines, psychiatry has had to struggle with handicaps which some of the younger branches of science, bacteriology for example, have been fortunate enough to escape. When attempts were made in comparatively recent times to establish psychiatry as a science, so many false theories and beliefs stood in the way, superstitions mainly, dating back to primitive times, that a century or more had to pass before it was possible to clear the way so that the scientific approach could manifest itself. Folklore, witchcraft, demonology and teleological and theological misconceptions have made the task of the builders of modern psychiatry vastly complicated and difficult. Dr. Lewis recognizes these influences and effects, and it is at this point that his philosophical view makes stimulating contributions to what in other hands might be a simple historical narration.

Because psychiatry, by its very nature and content, does not lend itself to experimental presentation, there are those who affirm that it is not a science. Lewis retorts that if that is the crucial test for science, geology, paleontology and several other branches of learning must be barred. As the author correctly points out, experimental procedures appropriate to investigation in some fields do not necessarily apply to all; but critical analysis and comparisons of accumulations of data when correctly interpreted will establish the truth or falsity of a thesis. This, after all, is the aim of scientific study. He sees, as the principal difference between modern

scientific investigation and older methods, experimentation, the proving of hypotheses and methods of verification, which distinguish present-day, fruitful scientific observations from the dogmas of ancient times when these checks and rigid controls did not exist. As a result, the modern experimenter avoids the pitfalls of misinterpretation and, consequently, of false hypotheses and conclusions which distinguished the earlier period. If, says Dr. Lewis, we are to make a true science of psychiatry, the experimental method should be adopted wherever it is applicable. It is obvious, however, that it can hardly be made applicable elsewhere than in organic reactions. When one seeks to understand the emotional disorders, other approaches will have to serve.

This book is the record of three public lectures given at the New York Academy of Medicine but it is more than the lectures. If one may have heard the oral presentation, he still will find inspiration in the opportunity to read the printed words.

Medicine and Human Welfare. By HENRY E. SIGERIST, M. D., D. LITT. 148 pages, with 20 illustrations and index of names. Cloth. Yale University Press. New Haven, 1941. Price $2.50.

Dr. Sigerist is William H. Welch professor of the history of medicine in The Johns Hopkins University. His present volume, based on the sixteenth series of Dwight H. Terry "Lectures on Religion in the Light of Science and Philosophy," is made possible, as he notes, by the fact that the deed of gift establishing the lectures "is conceived so broadly and in such a liberal spirit as to permit of any honest endeavor that is concerned with the improvement of human conditions." Readers of this little volume may well be thankful for that broadness and liberality, for Dr. Sigerist's principal subject begins to take leave of religion at about the point where the medicine man parted company with the priest-sorcerer. He pertinently remarks, however, that religion has never given up its pretentions to practise medicine. "In every man's life there comes a disease against which there is *nulla herba in ortis.* Where science has reached its limits, a miracle is hoped for."

Dr. Sigerist has divided his concise little summary into three parts, a section dealing with disease—its history, ancient theories concerning it, its modern prevalence and cost—a section treating in similar fashion the problems of personal hygiene and public health; and a section devoted to the history and the craft of the physician. It may be of interest to note his observations that in ancient Rome a physician was a slave who "brought a good price on the market, as much as a eunuch," and that the respected

modern medical man enters upon his scientific career with an oath he is "sure to break," a practice which, the author remarks, is "not harmless."

Dr. Sigerist is "convinced that medicine, like education, will ultimately become a public service in every civilized country." All trends, he holds, citing modern psychiatry among other developments to prove his point, are in that direction. The prospect is one which the author welcomes, and his book is a strong and well-presented argument that the establishment of medicine as a public service would benefit both public and profession. Since their own specialty is already so largely in the service of the state, his discussion is well worth the particular attention of psychiatrists. It is proper to add here that the style of the little volume is entertaining and that the controversial nature of its thesis has not destroyed readability. The illustrations, ranging from a psychotic masterpiece by Van Gogh to a wood cut of a medieval bathhouse with a bearded burgher in the rôle of Peeping Tom, are appropriate and enlivening.

From Thirty Years with Freud. By Theodor Reik. 241 pages. Cloth. Farrar & Rinehart, Inc. New York. 1940. Price $2.50.

To the admirers and followers of Freud, this book will be welcome. It is an intimate and personal account of the author's lifelong friendship with Freud in Vienna; and while no satisfactory biography of Freud has yet been written, except his own, this writing by his friend and disciple throws light upon the character and personality which—though sketchy—is welcome. It will be only after a lapse of time that a true, dispassionate and adequate estimate of the man and of his accomplishments can be prepared. Another generation must pass and the bitterness and vindictiveness of his enemies and critics must have been buried with them before a sound appraisal of the value of psychoanalysis to science can be set forth.

It is the fate of great thinkers that they are misunderstood and reviled. Charles Darwin is an example. He has been dead more than 50 years and the theory of evolution and of the descent of man has been accepted by men of science all over the world, but there are still groups, some of them large and important, which abhor the name of Darwin and even laws have been passed in some states of the Union that forbid teaching the truths of evolution. That was true because Darwin disturbed the complacency of the science and religion of his day, and Freud has done very much the same. It is intellectually unforgivable to overturn the long accepted errors of the teachers and theologians.

Though it would be futile to write a life of Freud at the present time, there are men living who could make valuable contributions. This book is one. A. A. Brill, Ernest Jones, Ferenczi and many others have a good

deal to contribute. All such should be asked to write what they can contribute and it should be stored in the archives of the International Psychoanalytic Association until the time arrives that a satisfactory biography and an estimate of Freud's life and accomplishments can be prepared.

In the meantime Dr. Reik's book will be found to be pleasant reading. Perhaps he idealizes his hero, but opinions aside, the plain factual statements deserve attention and throw light upon Freud, the kindly teacher, the conscientious scientist and good man. It is a book that should have a wide circulation among students of psychology and psychoanalysts. Others, perhaps, would not comprehend its full significance.

Artistic Behavior in the Abnormal. (A Survey of the Literature on) By ANNE ANASTASI AND JOHN P. FOLEY, JR. Psychological Monographs, Volume 52. Edited by John P. Dashiell. 71 pages. Paper binding. The American Psychological Association, Inc., Northwestern University, Evanston, Illinois, 1940. $6.00 per volume.

The present monograph is Number 6 of Volume 52 of the series issued by the American Psychological Association. The other monographs in the volume are: "Studies in the Psychology of the Deaf," "Preparatory Set (Expectancy)—Some Methods of Measurement," "Studies in Quantitive Psychology," "Reminiscence and Gestalt Theory," and "An Experimental Study of the Effects of Subliminal Stimuli."

This study seems to have been undertaken for the purpose of reaching a better understanding of the nature of artistic behavior and imaginative activity in general. The work was done under the auspices of the Columbia University Council for Research in the Social Sciences. It covers drawings and paintings, nature work, plastic art, music and other arts. For many decades, projects have been carried out in mental institutions for giving employment to patients under treatment, and artistic interests have usually predominated. This has been true, not only in America, but in the institutions of western Europe.

The authors have had no practical experience with the subject and the study is of psychiatric literature going back nearly 100 years. Many cases are reported in some detail, contrasting the production of artists prior to and subsequent to the onset of their mental disorders. In the case of music, observations are reported as having been made on musical sounds heard as hallucinations.

The pamphlet would be of interest to anyone who is investigating the artistic behavior of psychiatric patients and mentally deficient persons. No practical conclusions can be drawn from it. A bibliography comprising 227 references adds to the value of the publication.

Criminal Youth and the Borstal System. By WILLIAM HEALY, M. D. AND BENEDICT S. ALPER. 251 pages with selected bibliography and index. Cloth. The Commonwealth Fund. New York, 1941. Price $1.50.

That American reformatories reform few is a glittering generality which it is perfectly safe to accept—for it may be documented abundantly by anybody who takes the trouble to inspect any state's prison statistics. That Great Britain has developed a reformatory system which, by contrast, does reform great numbers is a fact less generally accepted in this country. The Borstal System, in process of experimentation and development for more than 40 years, seems to have been almost unnoticed by Americans. The bibliography which Dr. Healy and Mr. Alper selected for this book lists only one American and one Canadian title; and the authors have found only one American reformatory, the New Jersey reformatory at Annandale, in which there is important application of Borstal principles.

The present volume represents the third study of the Borstal System in which Dr. Healy has taken part. Unfortunately, it was interrupted by the war; but Mr. Alper was able to see enough of the more recent developments in a visit during the summer of 1939 to confirm his coauthor's earlier impressions of the system's worth. The Borstals, briefly, are institutions for training and supervision, for periods up to four years, of criminal youths between the ages of 16 and 23 who are certified as fit for treatment by the prison commissioners. The experiment began in a wing of Bedford Prison 40 years ago and developed to the point where in 1939 there were nine Borstal institutions, five walled or partly enclosed, four completely open.

Discipline is maintained by officers in civilian clothing; and the more personal supervision and what, in effect, is psychotherapy are directed by house masters and supervising officials selected from widely varying types and classes. It is interesting to note that the system is frankly adapted from the most aristocratic feature of English education, the public schools, and that a modification of the program intended to train the leaders of British public life is serving well to rehabilitate Britain's young criminals.

The present volume is necessarily compressed and lacking in detail. Nevertheless, it could serve very well as a hand book for a new experimental Borstal, and it deserves wide reading. The authors note that American and British crime statistics are not comparable; but, while they thus lack complete statistical demonstration of their thesis, they produce abundant evidence to testify to the superiority of the Borstal method in training for trades, in parole assistance and supervision, and in general acceptance and cooperation by the public at large.

Adaptation of the Borstal system to America under the model Youth Correction Authority Act which was recommended last year by the annual meeting of the American Law Institute is earnestly advocated by the authors. They suggest an improvement over the original system which would be of vast importance. Noting that "no small part of the work done with Borstal lads partakes of the nature of psychotherapy," the authors find that the "fine endeavor" to influence these youths is "based very largely on the subjective impressions of the individual members of the staffs." Psychiatrists, it is observed, are not at hand to undertake this work, and the staff members are not trained psychiatrically. Psychiatry is recognizing more and more that the problem of crime is within its province; and we may hope some day to see the experiment of an American Borstal under psychiatric direction. Such an experiment appears—on the evidence of this book—to be indicated.

Training and Efficiency. An Experiment in Physical and Economic Rehabilitation. By E. Jokl, E. H. Cluver, C. Goedvolk and T. W. de Jongh. 188 pages with reference, appendix of original data, 78 tables, 28 figures and 18 illustrations by E. Ullmann. Boards. Quarto. South African Institute for Medical Research. Johannesburg, South Africa.

This monograph is the second of a series of four which deal with research into a training and rehabilitation project which seems to have had astonishing success in the Union of South Africa. It is the Special Service Battalion, established some eight years ago to provide discipline and training for unemployed young men at the joint expense of the departments of defense and labor. Shortly after its establishment, South Africa was confronted with a situation where there was an excellent labor market, with a large number of European unemployed—a great proportion of whom seemed to be unemployable. The authors note that from 1933 to 1939, 13,815 boys were attested to the special battalion courses and that 10,753 completed their training. As proof of successful attainment of the course's objectives, it is noted that 9,409 of these subsequently found employment.

The authors of this study appear primarily interested in physical training, sports and public health; and they have approached the problem from that point of view. Instead of the conventional presentation of gross measurements based on averages of large groups, they made an intensive study of anthropometric, physiological and performance tests of 32 young men before and after their six months training and subjected their figures to a most minute statistical analysis. Within the group, they made quantitative

correlation studies of various sub-groups, such as those showing best and worst performances at the three mile run before and after training. In spite of the small numbers, their results, therefore, may lay claim to significance.

As might be expected, significant improvement was found in all classifications after conclusion of the course, which combined military, school and trade training. Interesting from the psychiatric point of view is the incidental finding by the authors of pronounced improvements in physiognomy—accompanied in at least one illustrated instance by disappearance of a marked convergent strabismus—improvements which must have had beneficial psychological as well as physiological effects. The authors stress the statement that they regard their anthropological and physiological results "mainly as measurable equivalents of a more profound and more complex transformation," in which they indicate psychological benefits are included. It is to be hoped that in their later reports they can devote more attention to this problem.

The present volume contains data which should be useful to all concerned with the medical aspects of military training. It is well printed and designed. Not the least of its attractions is the series of E. Ullmann's original sketches for the cover and 18 plates, depicting the boy from his days of school and slum home to rehabilitation through training.

The March of Medicine. NUMBER V OF THE NEW YORK ACADEMY OF MEDICINE LECTURES TO THE LAITY. 154 pages with index. Cloth. The Columbia University Press. New York, 1941. Price $2.00.

This fifth series of lectures to the laity, delivered by distinguished leaders in their specialized fields, presents, in the words of the introduction, views of six faces of the "multifaceted polished stone" which is medicine. The histories of chemotherapy, of research on the blood, of viruses and of bronchoscopy are presented by Perrin H. Long, Paul Reznikoff, Thomas M. Rivers, Chevalier Jackson and Chevalier L. Jackson.

Psychiatry, as was also the case in the fourth series of lectures, is well represented. "The Inheritance of Mental Disease" is discussed by Abraham Myerson in a lecture which should be "must" reading for non-medical campaigners for eugenics, a term which Dr. Myerson observes, "has become synonymous with sterilization to prevent the propagation of the unfit." Dr. Myerson says, in non-technical language, that the constitutional factors in the functional psychoses are not well understood and that in mental defect—where inheritance is more firmly established—the survival rate is so low that extensive sterilization measures would have little effect on the intelligence level of the general population.

Dr. Richard H. Hutchings delivered the other lecture on psychiatry, "The Ascent from Bedlam," a sketch of psychiatric history from the days of Hippocrates, with an outline of modern achievements in the field and an appeal for greater public understanding of and tolerance for the mentally ill. With its emphasis on modern methods of prevention and cure, this lecture might well be addressed to the vast multitude which still thinks of mental disorders in terms of "insane asylums," straight jackets, chains and hopeless misery.

The little volume in which these lectures are incorporated is in no sense a popular history of medicine. It is something which may be rather more valuable, an instrument by which the layman may gain insight into the tremendous progress and the manifold specialties of the science. Its chapters on psychiatry can be recommended for the lay reader without reservation.

Feeding Our Old Fashioned Children. By C. Anderson Aldrich and Mary M. Aldrich. 112 pages. Cloth. The Macmillan Company. New York. 1941. Price $1.75.

This little hand book is of interest to psychologists for the reason that it is almost the only work of its kind which stresses the emotional and psychological importance of breast feeding for the infant.

The authors have been interested in the welfare of children, particularly with reference to suitable diets for them, for a long time. This particular book is an outgrowth of an earlier brochure on "Cultivating the Child's Appetite." The text is written in language suitable to be understood by any educated person and when unusual or technical terms are necessarily used, they are suitably defined.

The Aldriches sensibly recognize the accomplishment of their aim is not simple, that no two children are identical as to their feeding problems and that each individual child must be studied until his difficulty is understood, whether it be capricious appetite or gastro-intestinal disorder.

It is a pleasure to note the recognition that the psychological and biological needs of the child are found to be closely related. The authors recognize, too, that the child draws something more than milk from its mother, that the act of nursing and the intimate contact with her gives an emotional satisfaction not to be gained in any other way and one which is important for his psychological development.

While one would like to see this good doctrine amplified, there is at least one good page that would redeem—if redemption were called for—many other pages of less psychological interest. The following is worth quoting for this significance:

"When a mother, for instance, picks up her screaming baby, cuddles him close, and allows him to nurse his fill, how does she support the needs of growth and make him more efficient?

"Let us see what she has accomplished by this simple act. The natural signal of his hunger cry has been respected; his hunger pains have been relieved; he has been warmed and made to feel welcome by his mother; he has had the sensory experiences of tasting, smelling, and feeling; he has used his nursing equipment, the rooting, suckling and swallowing reflexes; his appetite control has functioned; in fact, as far as eating goes, he has had a chance to exercise the whole gamut of power with which he is endowed at that time. What is more, he has found his work pleasant and has been satisfied in the process.

"When this experience is repeated over and over again, day after day, nursing becomes an eager delight to the child. The psychologists would say that he is 'positively conditioned' toward eating." (And, the psychologist would add, toward life and love.) "It is the repetition of this emotional feeling of pleasure that gradually raises the feeding act from a simple reflex into levels where the brain begins to function. Thus, the net result of his mother's satisfying response is a higher level of efficiency for him. This is the most practical kind of child care."

However, no apologies are necessary for this book; it is well gotten up, the illustrations are well chosen and attractive, and the index is ample.

EDITORIAL COMMENT

CAUSE OR EFFECT?

The question of the rôle of alcohol in mental disease is one to which at present we can, of course, give no full or completely satisfactory answer. Our concepts of the problem are in a state of flux and cannot be crystallized until there has been much more study and investigation and until new knowledge becomes available to science. Although a number of capable statisticians have devoted attention to the matter, no generally acceptable conclusions have been presented, either as to the extent of alcoholism or as to its influence in the etiology of the psychoses. Alcoholism is elusive of description and identification. Until there is a general acceptance of what we are talking about, it will continue to be idle to make up tables and draw conclusions as to its prevalence. Obviously the difficulty lies near the foundation upon which we must build any effort to study the problem. Do all men and women who consume alcoholic drinks identify themselves, thereby, as alcoholics? An affirmative answer would, manifestly, be absurd. When does a moderate drinker become an excessive drinker? Standards for classification are lacking.

THE PSYCHIATRIC QUARTERLY discussed this subject briefly in an editorial last April, protesting against what appeared to be undue emphasis in some quarters on the extent of alcoholic etiology of the psychoses; and Dr. Lawrence Kolb, assistant surgeon general of the United States, who is assigned to the Public Health Service's division of mental hygiene, brings the question up again in a letter commenting on that editorial. Saying that he had not, as a matter of fact, made an assertion which had been attributed to him —to the effect that more than one out of every 10 persons admitted to mental hospitals in the United States for the first time are victims of alcoholic psychoses or alcoholism—Dr. Kolb calls attention to United States census figures quoted in his paper, "Alcoholism and Public Health," which was read at the meeting of the American Association for the Advancement of Science in Philadelphia last winter. The official figures show, for certain recent years, total combined first admission rates for the alcoholic psychoses and for alcoholism without psychosis of more than 10 per cent for America's mental hospitals. "This," Dr. Kolb writes to THE QUARTERLY editor, "would justify the statement attributed to me." It should be noted that Dr. Kolb is interested here in all phases of the alcohol problem which relate to public health—not simply in psychiatry's concern with alcohol as a factor in mental disease. It should be observed also that these Census

Bureau figures are available for ready misuse by anybody who chooses to assert that they indicate alcohol's responsibility for a tenth of the psychoses which require hospitalization in this country—an assumption which, it is gratifying to note, Dr. Kolb makes nowhere in his "Alcoholism and Public Health" discussion.

Dr. Kolb remarks on the difficulty, which has just been discussed of determining the extent of alcoholism. He pertinently notes that the census figures he cites do not give a complete picture of the extent of hospitalization for alcoholism, since many states, New York among them, endeavor to exclude alcoholics without psychoses from their mental hospitals. For similar, as well as for additional, reasons, he comments that records of deaths due to alcohol, of hospitalization for alcoholic poisoning and of crime attributed to alcohol cannot be used to indicate the actual incidence of alcoholism in the general population. We may conclude that fundamental errors are inevitable here in determining who should be counted among the alcoholics. Intoxication is not a reportable disease. Who knows how many dipsomaniacs receive treatment in their own homes or in local hospitals for delirium tremens—which is a recognized psychosis?

There are other difficulties in obtaining accurate statistics even when the problem is narrowed from the general one of alcoholism to the restricted field of the alcoholic psychoses. A common experience in the reception wards of psychiatric hospitals is to have a patient admitted suffering from an acute alcoholic delirium, and to have this clear up a week later and disclose the presence of a well-defined manic-depressive psychosis.

The question of alcohol—because it has been made a moral issue—is, like religion, almost impossible to discuss objectively and dispassionately, even in scientific circles. The prohibitionist speaks of alcohol as if it were an animate, evil force, waiting, like a beast of prey in ambush, to drag unwary victims down to destruction. The economist and sociologist consider alcohol as if it were a commodity, like coffee or tobacco, which has value greater than some and less than some other commodities, and which has varying potentialities of revenue or loss for the state, and of benefit or harm for its citizens. General medicine may look upon alcohol as an agent serving various purposes, depending on the amount and circumstances of its administration—as a food, a stimulant, a useful antiseptic and astringent, a substitute for opiates in relief of pain, a toxin, a "habit-forming drug," or a social beverage in which prolonged overindulgence may lead to various nutritional and other organic disorders, including organic psychoses. Among psychiatrists, of course, are practitioners who share to some extent in all these widely varying points of view.

There would be general agreement among psychiatrists, however, even among those who condemn alcohol on moralistic grounds, that all these concepts of the nature of the agent can cast only a superficial light on the problem of alcoholism. Even the new, experimental evidence reported from the New York State Psychiatric Institute and Hospital that susceptibility to alcohol varies along lines suggestive of allergy can tell us little about why some persons use alcohol without abusing it and why others habitually overindulge in it. Certainly no approach to the problem from the point of view of morals, economics or general medicine has done more than tell us something about how alcoholism works. No such approach has explained why.

If there ever is to be a solution of the problem of alcoholism, it is suggested that it is imperative to conduct more intensive research designed to answer the question, "Why?" And it is submitted that the familiar concept of alcohol as a drug in which overindulgence requires further overindulgence and so *ad infinitum,* does not constitute a satisfactory answer to that question—for it is possible clinically to withdraw alcohol under controlled conditions and to enforce abstinence under such conditions, without untoward consequences. There is a growing conviction among many psychiatrists of widely varying schools that the why of alcoholism is not to be found in the nature of alcohol itself but is, rather, in the unstable personality associated with alcohol's abuse. This concept is directly opposed to the popular view that the alcohol is the cause of the unstable personality. Yet the belief that the cause must be sought in the personality may be held alike by the organicist who feels that the alcoholic has inherited a defective and unduly susceptible nervous system and by the Freudian who sees possible constitutional factors but who also sees the primary explanation for alcoholism in the development of the individual or in his family constellation. But if this belief is to be accepted—and, essentially, it is the belief that the alcoholic is a psychoneurotic who uses alcohol as self-therapy for relief of his symptoms, it will deprive most physicians of such easy methods of treatment as the attempt at legal prohibition of alcoholic beverages, or the commitment of the alcoholic to a hospital with the belief that he is cured when he submits to enforced withdrawal, or the attempt to frighten the alcoholic by telling him he is a constitutional inferior and will be poisoned by what other persons can consume harmlessly. There is no specific for the psychoneuroses; the workers with shock treatment, for instance, report far less success than with the psychoses; and if abuse of alcohol is evidence of an underlying psychoneurosis, the road to successful treatment is long and hard.

Yet if the problem of alcoholism is to be solved, it may be necessary that this long, hard road be traversed, that the underlying psychic causes—whether they are called anxiety states, fears, feelings of insecurity, inferiority complexes or infantile fixations—must be uncovered and treated. The effort must be made, too, to obtain a greater understanding of why these abnormalities arise—with a view to reducing their incidence in future generations. And if psychiatry is to follow this program successfully, the general medical profession and the public must be convinced that alcoholism is evidence of psychic illness, not the cause of psychic illness, that alcoholism is not moral turpitude, that it is not physical "slavery" to a drug, and that treatment is not to be found in reproaches, sermons, shamings and enforced deprivations. In this connection, it might be well to avoid inferences such as that of attributing to alcohol a determining part in the etiology of every psychosis where the anamnesis indicates overindulgence in alcohol—and until it is understood what overindulgence signifies. We should avoid supplying ammunition to fanatics intent upon frightening the public into reenacting prohibition through fear of national "insanity."

There should be reference here again to Dr. Kolb. "Hospitals for alcoholics," his paper declares, "could well be operated on the same plan as that of the two Federal hospitals for narcotic addicts. These hospitals have tended to take sin and punishment out of narcotic addiction and to place treatment, rehabilitation and research in the foreground. They have had success beyond expectations. . . ."

Whether Dr. Kolb would concur in the opinion that alcoholism may usually be regarded as evidence of self-therapy for mental disorder, we are unable to say; but he writes pertinently: "Man . . . likes to escape from unpleasant conditions and to have some means of acting childishly without being aware of it. Alcohol is the least harmful of any of the drugs by which he can achieve these results." And in discussing the possibility of a rational approach to the subject, he says, "Above all we need to know more about the fundamental causes of alcoholism and the nature of the physical and mental changes that make users more and more susceptible and less amenable to treatment."

Here, we should like to repeat with emphasis, "We need to know more about the fundamental causes. . . ." We have too long been handling the problem as if alcohol were a cause, not an effect. That is true, even of those of us who have been thoroughly convinced theoretically that the cause of alcoholism is not to be found in alcohol, that alcoholic excess is merely an effect.

NEWS AND COMMENT

DR. GILLESPIE TO GIVE SALMON LECTURES

Dr. R. D. Gillespie, chief psychiatrist for the British Royal Air Force, and one of his country's outstanding authorities on psychiatry and neurology, will deliver this year's Salmon Memorial Lectures, as a result of months of negotiations by the lecture committee with the British government. He will speak on "Psychoneuroses from the Standpoint of War Experience" on November 17, 18 and 19 at the New York Academy of Medicine building. The lectures are expected to give much basic information to American psychiatrists on the problem of civilian morale in war time, as well as on that of morale in the armed forces under the conditions of mechanized warfare.

Dr. Gillespie, in collaboration with Dr. D. K. Henderson of Edinburgh, a former Salmon lecturer, is author of a "Textbook of Psychiatry;" and he is also author of a volume on "Disorders in Sleep." Previous to his work with the RAF, he was lecturer in psychological medicine at Guys Hospital and Medical School in London and lecturer in psychopathology at the University of Aberdeen.

Following the Salmon Lectures, which will be the ninth of the series, Dr. Gillespie is to lecture in Chicago, Toronto and San Francisco. Dr. C. Charles Burlingame, chairman of the Salmon Committee, has issued a general invitation to members of the medical profession and their friends to attend the Salmon Lectures. Among those joining the Salmon Committee in its appeal to the British government to grant a leave to Dr. Gillespie for his American lectures, were Dr. James K. Hall of Richmond, Va., president of the American Psychiatric Association; Dr. Winfred Overholser of Washington, secretary of the American Psychiatric Association; Dr. C. Macfie Campbell of Boston, president of the American Board of Psychiatry and Neurology and former president of the American Psychiatric Association; and Dr. Louis J. Pollock of Chicago, president of the American Neurological Society.

ANNUAL SOCIAL WORK CONFERENCE

H. Beckett Lang, M. B., assistant commissioner of the New York State Department of Mental Hygiene, is chairman of the mental hygiene section of the New York State Conference of Social Work, which will conduct its

annual meeting in Buffalo, October 21 to 24. The section has outlined a program for the identifying and handling of the psychiatric problems which may be presented by the social workers of all fields who register at the conference. A number of institutes will be held which are expected to be of special value to those interested in mental hygiene problems.

HANS BERGER OF JENA IS DEAD

Hans Berger, who devoted a lifetime to studying the activities of the brain and whose crowning achievement was the development of the electroencephalograph, died in Jena, Germany, on July 7, 1941. He was 68.

Seeking to determine the organic reactions which might be considered the background of mental activity, Dr. Berger devoted years to studying such problems as cerebral circulation, brain temperature and trauma. The invention of the vacuum tube as an amplifier of electrical activity gave him his opportunity to return to work which he had started in his youth when instruments were not delicate enough for his purpose. The new research began in 1924 and was first reported to the world in 1929. The results were greeted—like those of many other important medical discoveries—with about as much derision as serious interest.

Today, not much more than a decade later, the scientific validity of Dr. Berger's brain wave discovery and the use of the instrument he developed are firmly established. By use of the electroencephalogram, pathological lesions of the cortex are discovered and localized; convulsive disorders are more readily diagnosed; and much promising research is being carried on into other conditions. The interpretation of the electroencephalogram is still subject to much dispute; and the conditions it reflects are obscure; but the value of Hans Berger's discovery and its promise for future research have long been beyond doubt.

QUEEN CHARLOTTE'S APPEALS FOR AID

Illustrating medical problems created by months of air-raids which have left Queen Charlotte's Maternity Hospital the only large one remaining in London, is an appeal for financial aid from America received by THE PSYCHIATRIC QUARTERLY. The hospital, unendowed and giving free care for each mother, estimates its costs at $80 for each maternity case with pediatric aid for five years. Part of the text of the appeal, sent to Americans by personal letter, is printed on the second advertising page of this issue of THE QUARTERLY.

DISCRETIONARY, NOT A RIGHT

Because of the presence in this country of so many refugee members of their profession, psychiatrists will be widely interested in the decision last July of the New York Court of Appeals that the New York State commissioner of education cannot be compelled to endorse a foreign medical license for practice within the State without examination, although such license may be granted at the commissioner's discretion. The lower courts had upheld the right of Dr. Otto Marburg, widely-known Austrian neurologist who is now clinical professor of neurology at Columbia University and a research neuropathologist at Montefiore Hospital, New York City, to obtain endorsement without examination. But, deciding on the appeal of Dr. Ernest E. Cole, commissioner of education, against the decision, Judge Finch, in writing the opinion of the State's highest court, declared: "Where the action of the appellants involved a refusal to exercise discretionary power, no order to compel the exercise of the power will lie, except in the case where it is clearly shown that the refusal is arbitary, unfair or capricious." Only four foreign licenses have been endorsed without examination since the power was established in 1918.

NEW COMPILATION ON CIVILIAN MENTAL HEALTH

The Military Mobilization Committee of the American Psychiatric Association, of which Dr. Harry A. Steckel, superintendent of the Syracuse Psychopathic Hospital, is chairman, announces a further compilation "for the use of the members of the Association who are being called upon to discuss or deal with problems of civilian mental health during the present period of stress." The subject is "Social Institutions During Periods of Stress," and copies may be obtained on request from the subcommittee chairman, Dr. D. Ewen Cameron, Albany Hospital, Albany, N. Y.

DR. EDWARD B. LANE IS DEAD

Dr. Edward B. Lane, widely-known Massachusetts psychiatrist, died on September 17 in Milton, Mass., at the age of 81. He was superintendent of the Boston Insane Hospital, now the Boston State Hospital, from 1897 to 1905, when he resigned to go into private practice. He was formerly on the staff of Harvard Medical School and was professor of mental diseases at Tufts Medical School for 25 years. He retired from practice at a private sanatorium for mental patients six years ago.

DEATH OF DR. RALPH E. CLOGHER, M. D., D. D. S.

Death last June 10 in Utica State Hospital of Dr. Ralph E. Clogher brought to a close an unusual 22-year career in the Department of Mental Hygiene, for Dr. Clogher had served on the staff of Utica State Hospital, both as physician and as dentist. At the time of his death, he had been senior dentist at Utica for 11 years. A graduate of the University of Pennsylvania Medical School, Dr. Clogher served at Utica as medical interne and assistant physician from 1910 to 1913, resigning to go into private practice in Utica. Following service with the Red Cross in France during the first World War, he believed that diminishing hearing would interfere with his medical practice and returned to the University of Pennsylvania where he took his degree in dentistry. He returned to the State service as dental interne at Utica in 1922 and remained with that hospital until his death. Dr. Clogher leaves a widow, the former Mary C. Philo of Utica, and a son, Roger. He was 55 years old.

NEW RESEARCH UNIT AT NORTHPORT

A neuropsychiatric research unit, with Dr. James H. Huddleston, senior physician, director, has been established at the Veterans' Administration Facility at Northport, N. Y. Dr. William J. Turner, also a senior physician, will supervise the laboratory activities, and the special staff includes a biochemist, statistical, laboratory, secretarial and stenographic assistants. The unit is to conduct clinical and laboratory research in connection with neuropsychiatric disabilities among veterans. Efforts to standardize diagnostic and therapeutic methods are to be made; and the unit is to conduct classes in neurology, psychiatry and neuropathology for medical officers of the Veterans' Administration who may be detailed for instruction. Operation is under the immediate supervision of Dr. Hugo Mella, chief, Postgraduate Instruction and Medical Research Division, Medical and Hospital Service of the Veterans' Administration in Washington.

NEUROLOGY AND PSYCHIATRY COURSES

The postgraduate courses in neurology and psychiatry which are given at the New York State Psychiatric Institute and Hospital in cooperation with the College of Physicians and Surgeons of Columbia University and the Neurological Institute of New York will be conducted this year from October 6 to December 12. The announcement notes that arrangements may be made for a special extensive course in neuropathology in which the major part of the time is spent in practical laboratory work at the Psychiatric Institute.

37 ARE ON SUPERINTENDENTS' LIST

Thirty-seven who took the written examination last May 24 for promotion to superintendent of the institutions under jurisdiction of the New York State Department of Mental Hygiene have been certified as having passed. Three candidates were disapproved, and two failed to pass the test. Those who received a rating of 85 or higher are:

Hugh Gregory, 89.19, Queens Village; H. A. LaBurt, 88.69, Wingdale; Armando Ferraro, 87.51, New York City; Arthur E. Soper, 87.21, Brentwood; Leo P. O'Donnell, 86.95, Brentwood; J. P. Kelleher, 86.86, Poughkeepsie; Chris Fletcher, 86.71, Buffalo; Sidney W. Bisgrove, 86.67, Marcy; A. M. Stanley, 86.57, Orangeburg; Newton J. T. Bigelow, 86.52, Brentwood; W. M. Pamphilon, 86.52, Willard; Arthur W. Pense, 85.44, Wassaic; Willard H. Veeder, 85.30, Rochester; Art. M. Phillips, 85.24, New York City; Gordon Priestman, 85.07, Kings Park; Reginald R. Steen, 85.06, Kings Park.

DR. ROBERT PERCY SMITH DIES

Robert Percy Smith, M. D., F. R. C. P., one of the outstanding figures of modern British psychiatry, died in England on June 4, 1941. He was 87 years old. At the age of 34, Dr. Smith became medical superintendent of Bethlem Royal Hospital, a position he held for 10 years before resigning to go into private consulting practice. He had been president of the Medico-Psychological Association of Great Britain and Ireland and of the Psychiatrical and Neurological Sections of the Royal Society of Medicine, and for many years he was editor of "Brain." Dr. Smith's personality was outstanding, and he was the subject of many anecdotes. It is said that when he was superintendent of Bethlem he saw and spoke with every patient every day and required all members of the medical staff to do the same.

DR. FOLKERS IS HONORED

Dr. Karl A. Folkers of the pharmaceutical firm of Merck and Company received the $1,000 award in pure chemistry of the chemical fraternity, Alpha Chi Sigma, at the annual meeting of the American Chemical Society in Atlantic City, N. J., on September 8, when he described in an address to the society the isolation of the active principles of the plant, *Erythrina Americana Mill,* which have effects similar to those of curare on administration to human subjects. It seems appropriate for THE QUARTERLY to extend its congratulations to Dr. Folkers, for one of the earliest reports of the therapeutic results of his research was printed in THE QUARTERLY for July, 1940. It was a paper on "The Prevention of Metrazol Fractures by Beta-Erythroidin Hydrochloride," written by S. R. Rosen, M. D., D. Ewen Cameron, M. D., and J. B. Ziegler, M. D.

INDEX TO VOLUME XV

AUTHOR INDEX

SUBJECT INDEX

BOOK REVIEWS LISTED BY AUTHORS

BOOK REVIEWS LISTED BY TITLES

THE PSYCHIATRIC QUARTERLY

OFFICIAL SCIENTIFIC ORGAN OF THE NEW YORK STATE DEPARTMENT OF MENTAL HYGIENE

Published at the State Hospitals P[illegible]
Utica State Hospital, Utica, N. Y.

Vol. 15 October, 1941

THE PSYCHIATRIC QUARTERLY
OCTOBER, 1941
Vol. 15 No. 4

TABLE OF CONTENTS

SCIENTIFIC CONTRIBUTIONS

SPECIAL DEPARTMENTS